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MILK-BORNE BRUCELLOSIS IN MINNESOTA

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THERE have been 3,074 cases of human brucellosis reported to the Minnesota State Department of Health in the ten-year period, 1940-1949. It is estimated that approximately 25 per cent of the reported cases in this state are due to the ingestion of raw dairy products, chiefly raw milk and cream.[5] Therefore it can be assumed that in approximately 767 of the cases reported during this period infection with Brucella organisms has occurred in this manner.

The presence of viable Brucella organisms in market milk, including milk from certified herds, has been demonstrated on numerous occasions. Outbreaks of human brucellosis due to *Brucella suis* in raw milk from cows have been reported by Beattie and Rice,[1] Horning,[4] and Borts.[2] Beattie and Rice state that outbreaks of brucellosis due to *Brucella abortus* (bovine) on raw milk routes are relatively uncommon, and that the occurrence of more than four or five cases on a single raw milk route is rare. Steele and Hastings[7] report an outbreak of twenty-eight cases of human brucellosis traceable to raw milk. *Brucella abortus* (bovine) was isolated from blood cultures from two of the cases. They state that this is the first large brucellosis epidemic due to *Brucella abortus* (bovine) that has been reported. Damon[3] reports the isolation of *Brucella melitensis* from milk of cows.

The Minnesota State Health Department records of cases of human brucellosis attributed to consumption of raw milk indicate that the source of raw milk usually is the herd on the patient's farm or herds belonging to friends or relatives.[5] However, during the ten-year period, 1940-1949, there were 450 reported cases of brucellosis in which the source suspected by the reporting physician was raw milk from a supply other than the herd on the patient's farm. Included in these 450 cases were ninety-six cases attributed to a common source in forty-one dairies. Twenty-eight dairies were the source of two cases each; nine dairies, three cases each; two dairies, four cases each, and one dairy, five cases. In most instances the cases were spread over a considerable period of time.

The outbreak of three known cases of brucellosis in patrons of a raw milk dairy is reported in more detail since *Brucella abortus* (bovine) was isolated from raw milk purchased at a store selling milk from the suspected supply (Table I).

Case	Age	Date of Onset	Agglutination with Brucella antigen	Blood Culture
1	65	8-15-49	(9-23-49) +1:1280	(9-23-49) No growth
2	25	1-50	(4-19-50) +1:1280 (5-2-50) +1:1280	(4-23-50) *Br. abortus* (bovine)
3	42	2-24-50	(3-4-50) +1:5120	(4-10-50) No growth

These three patients all had symptoms compatible with the diagnosis of acute brucellosis. The diagnosis was confirmed in Case 2 by the isolation of *Brucella abortus* (bovine) from a blood culture, and in Cases 1 and 3 by high agglutination titers with Brucella antigen. The possibility of sources of infection other than raw milk from the suspected supply was ruled out by history.

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The dairy owner was not co-operative but volunteered the information that he supplied approximately 400 quarts of raw milk daily to his patrons through retail and wholesale trade. Information from the State Live Stock Sanitary Board revealed that the suspected herd had been tested for Bang's disease in 1947. One "reactor" and three "suspects" were found, and the reactor was removed from the herd. At the time of the present investigation the owner refused to have his herd tested or to make arrangements for pasteurization of the milk.* A quart of the suspected raw milk labeled "Natural Milk" was purchased March 29 in the hope of obtaining irrefutable proof that the milk from the herd contained viable Brucella organisms. A sample of this milk examined by Dr. M. H. Roepke, Professor of Veterinary Medicine, University of Minnesota, showed a strongly positive ring test. A portion of the milk was also submitted to the Section of Medical Laboratories, Minnesota Department of Health. Two guinea pigs were inoculated subcutaneously on March 31, 1950, one with the sediment of the centrifuged specimen, the other with cream of the centrifuged specimen. The pigs were sacrificed on May 17, 1950, at which time agglutination was present 1:640 with Brucella antigen in blood from both animals. Cultures from the spleen of both pigs showed Gram-negative, non-motile organisms identified as *Brucella abortus* (bovine) on June 15, 1950.

The above report was sufficient evidence to justify the Live Stock Sanitary Board's placing a quarantine on the herd. This provided that no raw milk could be sold. The owner then arranged for Bang testing of the herd, which disclosed twenty-nine negative animals and two reactors. The reactors were removed from the herd and the quarantine was raised by the Live Stock Sanitary Board.

*As of July 1, 1950 the sale of raw milk in Minnesota is prohibited by law,[6] except as purchased for personal use at the dairy farm where it is produced.

Summary and Conclusions

An outbreak of three cases of human brucellosis traceable to a commercial raw milk supply is briefly reported. The causative organism, *Brucella abortus* (bovine), was isolated from the market supply of raw milk. The outbreak demonstrates the difficulty that is encountered from time to time by official agencies in obtaining compliance with good public health practice.

Although the cases of human brucellosis that are traceable to raw milk purchased from commercial raw milk dealers will be eliminated by the amended pasteurization law, the cases traceable to private sources of raw milk will not be decreased. For this reason, public health education must continue until universal pasteurization of milk is practiced, including home supplies and milk purchased by the consumer directly from the farm. Apparently, reduction in the number of cases of human brucellosis from all sources in Minnesota continues to be dependent on the reduction of brucellosis in the animal reservoir, namely the livestock population of the state.

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THE RIGHTS OF THE COMMUNITY

Every community is entitled to safe water, food, and milk, and protection from unsafe disposal of wastes; to as safe an environment as we know how to provide including pure air; safe streets, homes, places of work, and places of education and recreation; to the best protection we know how to provide from the contagious diseases, including tuberculosis and the venereal diseases; access to good medical care and hospitalization when needed; to the best protection we known how to provide against the special hazards of maternity and infancy; to the best facilities we know how to provide for the healthy development of our children, including correction of crippling physical and mental defects; recognition and treatment of rheumatic fever and other heart disease, and to the knowledge and facilities necessary to prevent as many deaths as possible from cancer, heart disease, diabetes, and the other degenerative diseases.—WILLIAM P. SHEPARD, M.D., *National Tuberculosis Association Bulletin*, October, 1949.

THE MANAGEMENT OF STATUS ASTHMATICUS

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STATUS ASTHMATICUS is an acute condition of severe, continuous asthma, unrelieved by injections of epinephrine even when repeated frequently and in increased dosage. When an acute asthmatic attack fails to subside and becomes refractory to the usual sympathomimetic medications which in the past gave relief, the dyspnea becomes more severe, the cough unproductive and the patient remains in a constant asthmatic state. The patient is critically ill and may die unless the attack of asthma is broken. When this occurs, its treatment taxes the ingenuity of the best clinician.

The above condition must not be confused with intractable asthma,[9] a chronic process usually associated with chronic bronchitis, bronchiectasis and pulmonary emphysema. These patients are usually difficult to control by the usual allergic management. The intervals between the acute paroxysms of asthma are seldom free of wheezing.

Status asthmaticus may occur in the acute extrinsic asthmatic as well as in the chronic intractable asthmatic. It occurs more frequently in the chronic intractable asthmatic (1) because of the difficulty in controlling the asthma adequately and (2) because the usual associated pulmonary pathology makes one more susceptible to bronchitic infection. This condition occurs less frequently in the extrinsic asthmatic and then it is usually due to specific sensitivities. When status asthmaticus occurs in the extrinsic asthmatic, the solution of the problem may be relatively simple, representing nothing more than the removal of the offending agent or agents from the patient's environment, or the removal of the patient from them.

Most of the fatalities in bronchial asthma occur in status asthmaticus. Death may be due to asphyxia or cardiac failure, but just as often, perhaps, it is due to exhaustion and dehydration.

The most consistent pathological finding in patients who have died in status asthmaticus[15] is the presence in the small, medium or large bronchi of thick, tenacious, gelatinous secretions which the patient was unable to raise. Much of the bronchial tree may be entirely occluded. In addition to these mucous plugs, edema of the bronchial walls and bronchospasm contribute to this bronchial occlusion.

When these patients are first seen, they give the classical picture[3] of severe asthma. They are usually in a sitting position with the body bending slightly forward from the waist, their hands grasping the edge of the bed or chair. The accessory muscles of respiration are forcibly in use, the face is drawn and ashen. The pallor may at times give way to cyanosis. Perspiration is profuse, for these patients are laboring for breath. Unconsciousness or disorientation may occur suddenly. The patient is terrified because of the repeated failure of the usual therapeutic medications. In addition, the family is anxious. There is a generalized spirit of hopelessness present and, not infrequently, the physician shares this feeling. This period of intense dyspnea may last from a few days to a week or two.

Examination of the chest elicits surprising findings to the uninitiated. Everyone is familiar with the "bandbox" heard in mild cases of asthma where there is only partial but wide-spread occlusion of the bronchi, as a result of which the sounds are widely distributed. In status asthmaticus, one finds areas of diminished and absent breath sounds, areas of quiet that to the experienced observer are ominous. In these patients, areas of the lungs have ceased to function normally because the bronchi serving those portions may be partially or totally occluded by thick, tenacious, gelatinous plugs of mucous. When one listens to such a chest, the need for prompt and vigorous therapeutic measures is immediately apparent.

The basic principles involved in a proper therapeutic approach in the treatment of status asthmaticus are (1) to increase the lumen of the respiratory passageway, and (2) to decrease the minute volume of respiration.

The following discussion sets forth a routine for the treatment of status asthmaticus which has produced the most favorable response.

Hospitalization.—Hospitalization should be insisted upon immediately. This will accomplish

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several indispensable purposes. First, the patient is removed from intimate contact with over-anxious relatives. Further, the hospital offers trained personnel, equipment and medicinal agents not readily available in the home.

Although most of the patients fall in the chronic intractable or infectious group, environmental factors should not be overlooked. Thus, the removal of the patient from the environment in which this condition developed may frequently be beneficial. If the status asthmaticus is due to pollen in the air, air conditioning with filtration is desirable if available. The ordinary precautions for the preparation of a dust-free room should be adhered to. It is wise to cover both the pillows and mattress with non-allergenic encasings. Flowers should be prohibited.

Reassurance.—As stated earlier, the patient is both terrified and panicky. This in turn will increase his exertional dyspnea. The patient fears that every breath may be his last. He must be reassured that the attack is controllable and that his cooperation is necessary to obtain results. A friendly, sympathetic and reassuring attitude on the part of the attending physician is a most vital and helpful therapeutic aid.

Cessation of all Epinephrine and Ephedrine Compounds.—We believe that this is the most important procedure employed. If nothing else can be done, this is the one thing to do. These patients have already received epinephrine, epinephrine-like, ephedrine and ephedrine-like compounds, to the point of nervous irritability and toxicity. They are "epinephrine-fast." Further epinephrine will only increase the patient's irritability and nervousness, produce tachycardia, palpitation, headache, pallor and weakness, with no effect on the dyspnea itself. The continuance of status asthmaticus proves the medication to have been ineffective, and a new start should be made. All sympathomimetic medications should be removed for a period of forty-eight to seventy-two hours, preferably the latter, and only then reintroduced. During this interval there is a strong temptation to reintroduce epinephrine, especially when the patient continues in relatively severe asthma and substitute therapy is of relatively little value. However, the discontinuance of epinephrine should be adhered to strongly during this interval.

When reintroduced, small quantities should be given, 0.3 to 0.5 c.c. (5 to 8 minims), and repeated as often as necessary, even within fifteen or thirty minutes. The smaller quantities will obviate the side effects of epinephrine and will produce the same therapeutic effect as larger quantities. It is preferable to use the aqueous (1-1000) epinephrine, rather than the prolonged type (1-500) in sesame oil, peanut oil, or gelatin. In a hospital there is no particular advantage in using the prolonged acting preparations for there is always the danger of overdosage from too rapid absorption, especially if the syringe is wet, with resultant side effects.

The problem in the treatment of status asthmaticus resolves itself into keeping the patient alive and as comfortable as possible for the next forty-eight to seventy-two hours following admission, for whatever the cause, when epinephrine is again introduced, invariably the patient will respond, especially if the other measures recommended in this paper are adhered to. If there is no accompanying infection in the bronchi, or the infection is minimal, the response will usually be immediate and fairly complete. If the accompanying infection is moderate, the response to epinephrine will be modified. The greater the accompanying infection, the less the response, but there will be a response. In the presence of infection, methods to combat the infection should be instituted immediately. The use of antibiotics, which are of great importance here, will be discussed subsequently.

Hydration.—This extremely important phase of treatment is almost always neglected. These patients are dehydrated.[12] They have been sick for a number of days without sleep, food or fluids. This is evidenced on admission by the very noticeable relative increase of the blood hemoglobin, red blood cell count, white blood cell count with a normal differential, along with a minimal increase of body temperature of about a degree. These soon return to normal after adequate hydration within twenty-four to forty-eight hours.

We routinely give 2 to 3 liters of 5 per cent glucose in distilled water and in isotonic sodium chloride solution alternately during the first two or three days of hospitalization. The addition of fluids will replace lost body water and bring about a positive water balance. They tend to thin out

the bronchial secretions and thus promote expectoration of the thick, gelatinous, inspissated mucous plugs in the bronchi. The dextrose used in hydration therapy will supply needed calories and replace liver glycogen, badly depleted because of the previous repeated injections of epinephrine and the failure of the patient to take adequate nourishment. Glaser[4] suggests that this depletion of glycogen may be a factor in the development of epinephrine fastness.

In the past, hypertonic dextrose[6,8] solutions up to 50 per cent, given in quantities from 50 to 100 c.c., at intervals of six to eight hours, have been recommended. The idea was to produce dehydration of the lungs and thus lessen the edema of the bronchi. However, its accompanying effect of dehydrating the patient generally and thickening the bronchial secretions defeated one of the major objectives of treatment—the evacuation of the thick, inspissated mucous plugs. Because of this effect, the use of hypertonic dextrose solutions should be discarded.

Aminophyllin (Theophylline with Ethylenediamine).—The bronchodilating effect of aminophyllin intravenously at times is life-saving. Initially, the patient should receive 0.25 gram (3¾ grains) in 10 c.c. of diluent given slowly, preferably through a fine needle. If this dose is sufficient for symptomatic relief, it can be repeated every four to six hours. If relief is only partial, the dosage may be increased to 0.5 gram (7½ grains) in 20 c.c. of diluent. When given slowly and regulated to the patient's tolerance, the toxic effects of aminophyllin, such as vertigo, faintness, headache, tachycardia, palpitation, extreme flushing and sense of heat, substernal distress, and nausea and vomiting may be obviated. If they do occur, they may be minimal. In uncomplicated asthma, aminophyllin is not a dangerous drug. However, in the presence of cardiac complications caution must be used. The need for repeated intravenous injections of aminophyllin may be lessened by inserting 0.5 gram of aminophyllin per liter of fluid during the period of venoclysis.

The drug is also moderately effective when given rectally, either in suppository form or as a retention enema. The suppository contains 0.5 gram of aminophyllin. One-half gram of aminophyllin powder dissolved in 30 to 60 c.c. of tap water may be used as a retention enema. Given in this manner, it can be repeated every 6 to 8 hours.

Continuous intravenous aminophyllin in status asthmaticus has recently been introduced by Goodall and Unger.[5] Dosage consisted of up to 2 or 3 grams of aminophyllin dissolved in 2,000 c.c. of 5 per cent glucose in physiological salt solution or distilled water. The solution is given continuously over a twenty-four-hour period for several days until relief is afforded.

We see no particular advantage to this method, because the same coverage can be achieved by employing repeated intravenous injections of aminophyllin togther with rectal suppositories or retention enemas, without the extreme inconvenience to the patient of having a needle in his vein continuously for three or four days. This is extremely important when considering that the patient in status asthmaticus is already in extreme discomfort because of his marked dyspnea.

Occasionally patients may become refractory to the intravenous administration of aminophyllin. Recently Prigal[10] has recommended the aerosolization of aminophyllin when this occurs. The contents of a 10 c.c. (0.25 gram) or 20 c.c. (0.5 gram) ampule are nebulized at six- to eight-hour intervals. We have employed this procedure in a limited number of patients. Definitive judgment as to its relative value remains to be determined.

As in the case of "epinephrine fastness," when patients become refractory to aminophyllin by intraveous injection or aerosolization, its use should be discontinued, as further dosage will serve only to increase its toxic effects.

The use of intravenous aminophyllin in the treatment of children may be employed in the same manner, the dosage being .006 gram per kgm. (1/20 grain per pound).

Inhalation Therapy.—Inhalation therapy is directed toward decreasing the minute volume of respiration. It rarely of itself will interrupt status asthmaticus, one must be extremely careful to make the patient more comfortable by diminishing the extreme respiratory effort caused by the anoxia, by enriching the surrounding air with oxygen.

Oxygen may be employed with a tent, nasal catheter, or B.L.B. mask. At times, patients will rebel against the use of a tent because of a feeling of claustrophobia. This may increase their

anxiety and nervousness, with resultant increase of their exertional dyspnea.

Barach[1] introduced a mixture of 80 per cent helium and 20 per cent oxygen, a mixture which has one-third the density of air. It therefore should diffuse more readily through the partially obstructed bronchioles. Its cost, however, is a limiting factor and, in our personal experience, oxygen has been equally as good.

Sedation.—In employing sedation, one must guard against over-sedation. However, measures to insure sleep and to overcome nervous tension are very necessary. We have used Demerol repeatedly, but with considerable caution. Used judiciously, it has proven to be a most effective drug. Its action[2] has apparently been twofold, sedation and a direct bronchodilating effect. In status asthmaticus, one must be extremely careful about respiratory depression and depression of the cough reflex, effects which are relatively minimal with Demerol as compared to the opiates.

In this connection, mention should be made concerning the use of morphine. In the past it has been used extensively, occasionally beneficially. However, one can say it should never be used in asthma, and especially so in status asthmaticus, where the patient is anoxic, exhausted and battling for life. Morphine depresses the respiratory center, diminishes the cough reflex and dries the bronchial secretions (especially if given with atropine). Thus, morphine actually promotes further anoxia—to the point of asphyxia—which is the very thing we are trying to combat. Because of the stagnation of the bronchial mucous plugs, the patient literally drowns in his own bronchial secretions. Vaughan[14] and Lamson[7] have shown that in many deaths due to asthma during status asthmaticus, morphine was given prior to death. The use of all other opiate derivatives should also be avoided.

The dosage of Demerol should be regulated with extreme care. Adults should never be given an initial dose exceeding 50 mgm. intramuscularly. It may later be necessary to increase to 75 mgm., and only rarely to 100 mgm. This can be repeated at six- to eight-hour intervals. It should be used for relatively short periods, three, four or five days, because of the possibility of addiction.[14] The routine use of Demerol for the relief of the usual acute attacks of bronchial asthma, as has been advocated, is to be condemned because of its properties of addiction. When using Demerol we have avoided using other sedatives, because of the possibility of over-sedation and the depression of all body functions.

Other sedative measures have been advocated by others.[15] Our experience with them is limited, but we will mention them briefly.

(1) Paraldehyde may be given rectally, 15 c.c. in 100 c.c. of olive oil at twelve-hour intervals.

2. Barbiturates may be given at four- to eight-hour intervals.

3. Chloral hydrate, 1 gram, and sodium bromide, 4 grams, may be given at four-hour intervals until the patient becomes drowsy; then stop.

4. A mixture of ether, 2 oz., and olive oil, 4 oz., mixed thoroughly, may be administered as a retention enema.

If any of the above are employed, only one should be used and not a combination. If used properly and carefully, sedation is extremely beneficial and life-saving. Its drastic use in an already exhausted and anoxic individual may be dangerous and disastrous.

Expectorants.—Methods which will thin out bronchial secretions and thus will help clear the bronchi of their mucous plugs are highly desirable. The best medication to achieve this is potassium iodide. It has been shown by Tuft[13] that the iodides are excreted in the bronchi in high concentration. Ten to fifteen drops of a saturated solution of potassium iodide taken orally are recommended four times daily until the patient is free of expectoration. If the patient is unable to take the drug orally, it may be given intravenously as sodium iodide. One gram may be added to a liter of the solution for intravenous administration by the drip method. If there is an intolerance to potassium iodide, enteric coated ammonium chloride tablets in 0.5 gram doses may be given four times daily.

In children, emetic doses of ipecac will produce forceful emesis and with it expectoration of mucous and clearing of the bronchial tree.

Manual Elevation of the Diaphragm.—In the presence of status asthmaticus physiological pulmonary emphysema is present. There is trapped air because of the partially and completely occluded bronchioles. Manual elevation of the di-

aphragm, as suggested by Gay,[2] is often followed by subjective relief as well as an increase in the vital capacity from 200 to 1000 c.c. The procedure is carried out as follows: the palm of either hand is placed underneath the ribs on one side and pushed upward and inward during the latter half of expiration. Then this is repeated on the other side. The escape of trapped air may frequently be heard as a wheeze. This procedure should be repeated three to four times daily.

Bronchoscopy.—Although we have not had occasion to use bronchoscopy, its use should not be overlooked. The mechanical removal of thick, tenacious mucus from the bronchi would appear to be a most reasonable treatment. Bronchoscopy has undoubtedly been restricted in its use because patients seem so gravely ill that any procedure which places a greater strain upon them would almost appear to be inadvisable. In skilled hands it is a relatively safe procedure and the risk is much less than that of possible asphyxia from the disease. However, preoperative medication should be kept at a minimum. Morphine and opiate derivatives are definitely to be avoided.

Antibiotic Therapy.—With the advent of antibiotic therapy, another powerful weapon has been added. As stated earlier, most patients in status asthmaticus belong in the chronic intractable or infectious group. Frequently an accompanying infection of the bronchi has been the cause of the status asthmaticus. The presence of infection is noted clinically by an increase in body temperature, elevated sedimentation rate, the presence of muco-purulent or purulent sputum, and leucocytosis with an increase in the polymorphonuclears.

Our routine is to use combined parenteral and aerosol penicillin therapy, so that the penicillin may reach the more superficial and deeper lying tissues of the bronchi in high concentration. Fifty thousand units of penicillin in 1 c.c. of distilled water, to which 3 or 4 drops of glycerin are added to stabilize the aerosol, are nebulized every three hours, with a six-hour interval during the sleeping hours. If the penicillin aerosol is to be continued after the epinephrine-fastness has been broken, it is advisable to precede the inhalation of penicillin by the inhalation of a few breaths of 1:100 epinephrine, or 1:200 isuprel, so as to widen the lumen of the lung. Very often, this therapy will have to be prolonged for five to ten days following responsiveness to epinephrine until the patient's bronchial secretions are free of discoloration and are at a minimum. At the same time, penicillin is administered parenterally with daily injections of 300,000 units of prolonged acting penicillin.

Because of the possible toxic effects of streptomycin and dihydrostreptomycin, its routine or combined use with penicillin is initially avoided. It is added only when the sputum remains purulent or in the presence of pencillin-resistant organisms in the sputum. Dihydrostreptomycin, because of its lower incidence of toxic effects, is then given by aerosolization in seven divided doses of 1 c.c. each per twenty-four-hour period in a similar manner as penicillin. The total dose per day ranges from 0.5 gm. to 1.5 gm. Its parenteral use is withheld. In our experience it has rarely been necessary to use streptomycin or dihydrostreptomycin.

As yet we have had no opportunity to use bacitracin aerosol, used successfully by Prigal,[11] and the newer orally administered antibiotics, aureomycin, chloromycetin, and terramycin.

Antihistaminics.—The recently introduced antihistaminic drugs are of little or no value in this condition. In fact, they are contraindicated, as they possess an atropine-like effect in drying up bronchial secretions, and thus aid in producing mucous plugs. Before substituting these medications, the action of which is neither so certain nor so prolonged, it is well to remember that epinephrine and epinephrine-like compounds are the most powerful antihistaminic agents now in use.

In summary, when one is confronted with a patient in status asthmaticus, the danger of death is ever present. The judicious use of the above procedures may be lifesaving.

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THE ROENTGEN DIAGNOSIS OF SILICOSIS

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I AM GRATEFUL for the honor and conscious of the responsibility of speaking to you on this occasion, a period dedicated to the memory of Russell D. Carman. This is not the time for me to attempt to refresh your minds concerning Dr. Carman's contributions to medicine. Many of these are a matter of record. Dr. Carman was a physician, scientist and teacher. He possessed such qualities as understanding, honesty, reason and justice, which were a source of inspiration and satisfaction to those of us who were privileged to know him. Honorary lectures such as this provide an opportunity for all to pause and contemplate the achievements of one of our great physicians, an opportunity for us to rededicate our lives to those things that will stimulate our greatest efforts toward improving medicine and through it providing greater service to our fellow man.

The subject which I have chosen for presentation is one of timely interest, although some of the symptoms and changes in the lungs produced by inhalation of dust in certain occupations were described by medical writers many centuries ago.

Our ideas concerning the prevention and control of disease have undergone a remarkable transformation during the past fifty years. The modern doctor, according to the late David Riesman, is no longer being called upon to treat illnesses which have disappeared or are rapidly vanishing. His work is to take on new orientation, that of guardian of the health rather than curer of ills for after all, "to guard is better than to heal; a shield is better than the spear."

Today in this industrial age, as never before, many of the forms of occupational disease fall within the province of the family doctor as well as the industrial physician. Our task is to be aware of the hazards affecting our working people in order that we may help make the industrial population a source of strength and not a source of weakness. We have to insure that as a result of modern industry and commercial procedure and environment, we do not saddle ourselves with a number of disintegrated and therefore unhappy, discontented men and women.

One of the earlier references concerned with dangers of dust exposure is Pliny's[61] description of the devices used by refiners to prevent inhalation of the "fatal dust."

In 1556, Agricola[1] described the perils of mining and the pestilential air breathed by miners. Ramazzini[63] in 1700 called attention to the possible relationship between dust inhalation and consumption. In the Renaissance, physicians and mining engineers were aware that the metal miners suffered from shortness of breath and died prematurely. Anatomists had described "heaps of sand" in the lungs of stonecutters and they called the condition phthisis. Thackrah,[74] in 1831, noted that sandstone workers died before forty, but there was no unusual instance of lung diseases in brick and limestone workers. Although the effect of various dusts in the lungs was recognized previously by other writers, Zenker[84] in 1867 is given credit for having coined the word "pneumonokoniosis." Shortly thereafter, in 1870, Visconti[77] described a pathological condition of the lungs resulting from inhalation of silica which he called "silicosis." In more recent years, numerous studies and investigations have been carried out in many countries. In the United States, the investigations carried on at the Saranac Laboratory under the direction of the late LeRoy U. Gardner[25] have added tremendously to our knowledge of the development of pneumonoconiosis and silicosis in the experimental animal.

Pneumoconiosis

Pneumoconiosis is a broad generic term used to describe all forms of pulmonary reaction to dust lodging within the lungs, with no implication as to character, severity, or effect on function. Certain of these reactions may be demonstrated by a roentgen examination of the chest, but in most instances they are entirely non-specific, are unaccompanied by formation of progressive fibrosis, and are of no clinical significance. In the light

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of present knowledge, however, we recognize at least two clinically important specific pneumoconioses, namely, silicosis and asbestosis, as well as a number of benign pneumoconioses resulting from the inhalation of a variety of inert but radiopaque dusts. The former may be productive of disability, whereas the latter are of clinical significance only because they may lead to errors in diagnosis through their ability to produce upon the roentgenogram a nodular pattern at times indistinguishable from that occurring in silicosis.

Silicosis

Silicosis is a form of pneumoconiosis which is thought to be due to the specific action upon the lung tissue of chemically free silicon dioxide in finely divided form and which produces changes that can be demonstrated in many instances on a roentgenogram. It is probable that all dusts, with the exception of chrome, free silica and certain silicates, produce very few changes in the lung that can be recognized with assurance on the roentgenogram.

In order to produce lung changes it is necessary for the silica dust to reach the lung alveoli, to get into the interstitial tissues either through the agency of macrophages (dust cells) or by way of alveolar pores, and to be deposited in sufficient concentration at some spot where either through its inherent physicochemical properties or together with some other factor or factors, as yet unknown, an abnormal amount of fibrous tissue formation may be stimulated.

In the early days, the tissue reaction to quartz dust was considered to be due to the sharp-pointed particles. This concept was invalidated when diamond dust and carborundum were shown to be benign. Then came the solubility theory of silicosis. This concept considers silica as slightly soluble, which in turn stimulates the formation of fibrous tissue. Although this theory has served as an excellent working hypothesis, several inconsistencies have been pointed out by Evans and Kascht.[20] These include:

1. A local concentration of ions, or a dissolution of the dust deposit has not been demonstrated.
2. Several benign silicates supply a greater number of soluble ions to the local tissue than does quartz.
3. There does not appear to be a constant relationship between solubility and the degree of fibrous tissue formation.
4. Silica in solution in the tissues would be precipitated as sodium silicate, which is benign.
5. It would appear that a fibrotic nodule would offer little functional defense to a substance in solution, as diffusion would progress through such a barrier.

In view of the above objections, Evans and Kascht[20] recently have examined various substances commonly responsible for the production of the pneumoconioses. They failed to find anything in the nature of a common chemical or physical property, such as solubility, hardness, sharpness, or known chemical reactivity, in the tissues that could explain the production of the characteristic fibrotic nodule. They found that the dusts known to produce fibrosis were composed of substances whose most stable form was that of an asymmetric crystal, and therefore, potentially piezoelectric. The benign dusts were either amorphous or of crystalline classes which are not piezoelectric.

Piezoelectricity is defined as being that property possessed by certain asymmetric crystals which allows a transformation of energy, in either direction, mechanical and electrical energy, Evans and Kascht.[20] If such a crystal is distorted by pressure, an electric polarity is produced, and conversely, if such a crystal is placed in the proper electric field, a distortion of the crystal surfaces is produced. There may be many related secondary effects. Evans and his associates have carried out a number of experiments which seem to support the thesis that asymmetric crystalline crystals under certain circumstances produce fibrosis.

Although many problems present themselves for future clarification, Evans and his co-workers,[19,21,22] have shown the following:

1. The ability of foreign materials to produce fibrogenesis is correlated with their crystalline structure.
2. Those tested amorphous materials and materials whose crystalline state possesses a central point of molecular symmetry (symmetrical crystals) are nonfibrogenic.
3. Those tested crystals which do not possess a central point of symmetry (asymmetrical crystals) and possess piezoelectric properties are fibrogenic.
4. Several materials previously untested bio-

logically, selected for their physical properties, were tested and shown to be fibrogenic.

5. Fibrous tissue reactions are considered to be stimulated by releases of energy in mechanical or electrical states within tissue.

Parmeggiani,[51] in 1947, writing on the same subject states that since silica nodules also are formed in organs free from any mechanical activity, such as the spleen, liver, and lymphatic glands, it is not possible to explain the tissue damage by piezoelectric action due to pressure on the crystals.

I am not well enough informed about the biological effect of piezoelectricity on tissues to have an opinion as to whether the theory being tested by Evans and his co-workers is correct, but I enthusiastically believe that their investigations should be encouraged and supported. If their present concepts prove to be right, many of the baffling problems concerned with the control of silicosis may be explained and some of them solved.

Pathogenesis

Although it cannot be stated with certainty whether the effect theory is responsible for the tissue responses in silicosis, chronic pulmonary disease of the type under consideration is dependent upon an adequate concentration of dust exposure maintained for a sufficient period of time. The rate of development will be influenced by the adequacy of the drainage system of the lung.

The process by which the phagocytes and macrophages carry off the dust particles has been well described by Gardner[30,32] and others.

Inhaled dust first exerts its influence on living cells primarily within the bodies of the alveolar phagocytes, and it is here that fundamental differences due to the physicochemical composition of the irritant become evident. Inert substances, in which category the great majority of dusts belong, provoke no structural changes within the cells. In contradistinction, free silica exerts a specific effect. Degenerative changes, easily confused with those in the "epithelioid" cells of tuberculosis, quickly become evident. The enlarged cells contain visible lipoid. Their nuclei repeatedly divide, and giant cells comparable to the Langhans' giant cells of tuberculosis are formed. Eventually these migrating phagocytes concentrate the silica in and about the pulmonary lymphatics, where the toxic particles, either directly or indirectly stimulate connective-tissue proliferation, with the resultant formation of microscopic silicotic nodules situated in the immediate vicinity of the lymphatic trunks.

At this stage the only general influence of the disease thus far discovered is the increased likelihood that the more advanced changes of silicosis will result from the continued inhalation of silica-laden dust. After sufficient reaction has developed in the lymphoid tissues, the flow of lymph is retarded. The alveolar phagocytes, however, continue to take up fresh particles of dust but, due to their apparent inability to enter the lymphatic vessels, collect upon the walls of the air spaces proper. Once again, the silica exerts its specific effect, causing proliferation of connective tissue and the formation of parenchymal silicotic nodules. Microscopically the nodules are seen to be composed of hyaline collagen fibers and are evenly distributed throughout the lungs (Gardner.[32]) Such, in brief, is the pathology of simple silicosis.

Tissue reactions occur along the lymphatics accompanying the pulmonary vessels and bronchi, within the interstitial tissues and along the pleura.

From an examination of the mineral particles which have gained access to the lung it seems that the protective mechanisms of the respiratory tract exclude most of the particular material greater than 10 microns in diameter. Likewise it is accepted generally that the smaller silica particles, 0.5 to 2.5 microns in size, are more likely to produce lung damage than are the larger particles. It is believed by some investigators that certain dusts (diluents or contaminating dusts) may have modifying effects upon the action of free silica in the lung tissues; for instance, carborundum and aluminum oxide may retard and alkaline soap powders may accelerate the usual reaction to free silica. There is some uncertainty about this hypothesis, however, as Gardner[30] says that "definite proof of accelerators to the action of silica is yet lacking; it has been suggested that cases of 'rapid silicosis' in human beings may be due to excessive exposures to silica or unusual fineness with or without associated infection." It is evident, therefore, that in order to evaluate a dust hazard one must have a knowledge of the behavior of the dust in the course of its production and while it is suspended in the air, and its behavior in the respiratory system, as well as an understanding

of its specific action in the lung tissue (Hatch.[37]) *The knowledge that a workman is exposed to dust released in the processing of a material containing free silicon dioxide is insufficient evidence in itself to justify one in stating that the atmosphere at the breathing level contains harmful quantities of free silica dust.*

The reasons for this observation are supported by ventilation and sanitary engineers familiar with many of the differences in the behavior of dusts and some of the factors concerned may be abstracted as follows:[37]

1. The composition of the air borne dust may differ from that of the parent material.
2. The composition of the dust retained in the deeper portions of the lung may differ from that of the air borne dust.
3. The amount of dust retained in the respiratory system and the relative amounts retained in the upper portion of the tract and that found in the alveoli vary with different dusts.
4. The particle size of the alveoli dust differs from that of the air borne material.
5. The flocculating properties of dust vary and this affects the retention in the respiratory tract, its penetration to the alveoli, and the behavior of phagocytes toward the dust.
6. The rate of phagocytosis varies from one dust to another.
7. The toxicity of silica is apparently reduced in the presence of certain other materials.

According to Hatch[37] the relative significance of these several aspects of dust behavior is not fully understood but their importance has been demonstrated both in the laboratory and in industrial experience.

The modifications occurring from the inhalation of dusts containing mixtures of free silica and other minerals may be quickly passed. The primary pathology is the same, the essential differences being simply the result of co-existing silicosis and a benign pneumoconiosis, each of which alters to some extent the appearance of the other. It should be recalled, however, that some inert dusts, when mixed with free silica, cause varying degrees of retardation in the development of the silicotic process. At this time the use of aluminum deserves brief but special mention. Denny, Robson, and Irwin[14] were the first to announce the specific inhibitory effect of aluminum. Their animal experiments and those of others,[33] some of which were conducted simultaneously in another laboratory, have demonstrated conclusively that metallic aluminum and aluminum hydrate, when given by inhalation, will prevent the fibrous reaction to quartz. The possible clinical applications of such a discovery are obvious, and have already stimulated considerable investigative work from the standpoint both of prophylaxis and of therapeusis. While a complete discussion of their present status is beyond the scope of this paper, it may be said that further evaluation is required and that aluminum therapy should in no circumstances be applied as a substitute for other and already recognized methods of dust control.

As evidence of the necessity for caution, are the lung changes associated with the manufacture of alumina abrasives reported by Shaver,[72] and the experimental observations of Evans and Zeit[21] on aluminum phosphate.

The alteration of the silicotic process when complicated by the presence of infection is not nearly so simple.[58] The increased susceptibility to tuberculosis in man recorded by Merewether,[48] and proved by the classic animal experiments of Gardner,[25,28] needs no recapitulation. Two facts, however, must again be emphasized: (1) that it is the presence of associated infection which accounts for most of the disability arising from silicosis, and (2) that infection, when it occurs, may manifest itself in either of two ways, namely, by the development of tuberculo-silicosis or of silicosis with tuberculosis.

Tuberculo-silicosis is common and according to Brumfiel and Gardner,[6] whose observations are adequately substantiated both clinically and pathologically, it is "a distinct disease entity with certain characteristics peculiarly its own, in that it is neither silicosis nor tuberculosis nor is it a simple summation of the two." It is the result of the interaction of tubercle bacilli and silica in the same area, with the resultant formation of tuberculous granulation tissue together with a modified type of silicotic reaction. Pathologically, it is characterized by the formation of slowly developing, well defined, hard or rubber-like areas of massive conglomerate fibrosis surrounded by a marked degree of emphysema. On microscopic section, nodules are found embedded within dense hyaline fibrous tissue which virtually obliterates the normal pulmonary structures. The tuberculous component of the process sometimes is identified

easily by the presence of widespread caseation, but not infrequently it is only after painstaking search that isolated, non-caseous tubercles or occasional clumps of acid-fast bacilli are discovered.

Silicosis with tuberculosis, by comparison to tuberculo-silicosis, is rare, but it does occur in one of two forms: either (1) as the result of infection superimposed upon a progressive and still active silicosis or (2) as the result of infection superimposed upon an old and already stabilized silicotic process.

In the first instance the infection becomes acute, and its course is usually one of uncontrollable extension. In the second, tuberculosis develops upon a background of an already stabilized silicosis in which the quartz particles are presumably completely isolated within their fibrous nodules and thus exert no effect upon the superimposed infection. We have then simply the coexistence of silicosis and tuberculosis within the same individual, but without modification or acceleration of either disease process by the other.

The effect of silicosis upon non-tuberculous infection is less well documented. It cannot be denied with finality that non-specific inflammatory reactions within silicotic lungs occasionally may be the precursors of conglomerate areas of fibrosis. Proof, however, is lacking, and the meager available evidence is opposed to such an assumption. Pierpont[60] has demonstrated that in his iron-mining area the incidence of pneumonia is no greater than in the general population, and that the behavior of the disease when it does occur is unaltered by an underlying silicosis. Similarly in rabbits it has been shown that silicosis exerts no effect upon their susceptibility to infection with Type III pneumococcus.[78]

On the other hand, Gardner,[32] after microscopic examination, was able to discover in only 60 per cent of cases showing massive conglomerate fibrosis, indisputable evidence of tuberculous infection. Perhaps in most if not all of the remainder, the silicotic fibrosis had obscured the tuberculous component of the process, and it was his feeling that an underlying tuberculosis was the etiologic factor accounting for at least the majority of massive fibrous lesions.

Roentgen Considerations[55]

It is generally conceded that the roentgenographic examination, properly done, is the most precise method at our command for demonstrating pathological changes produced by pneumoconiosis or silicosis in the living individual. There are, however, many other conditions that produce shadows in the roentgenogram which may simulate some of the various shadows found in pneumoconiosis.[54] In order to evaluate correctly the various shadows observed in a roentgen study of the chest, it is necessary for one to possess some knowledge and experience concerning such an examination, and certain information about the patient being studied. Some of the more important requirements may be enumerated as follows:

1. A knowledge of the anatomy of the chest and some of the physiological manifestations of the various structures contained therein; an understanding of the histology of the lungs and of their lymphatic system.
2. A thorough familiarity with roentgenoscopic and roentgenographic appearances of the normal structures of the chest and their permissible variations.
3. A clear perception of the pathology of pneumoconiosis and of lesions that give a somewhat similar roentgenographic appearance.
4. Some knowledge of the history of the individual, especially the occupational record and familiarity with the physical signs in the particular patient.
5. Some information concerning the industrial process that is responsible for the production of the dust. Dust counts at breathing levels and chemical analyses of the dust are exceedingly important when available.

If all of the above information is available, one should be able to render a diagnosis which although presumptive is likely to be correct in the majority of instances. Very often, however, one gives an opinion on insufficient data and in so doing referring physicians become confused and injustices occur.

I have always regarded the roentgen examination as a consultation, and used as such it is likely to be more valuable. An ideal program for the diagnosis of pneumoconiosis in the living would include a study by a group, the members of which would be a general physician, a rhinologist, a bronchologist, a clinical pathologist, a specialist in tuberculosis, a physiologist interested in pul-

monary function, an engineer expert in industrial hygiene and a general roentgenologist. Such a program is ambitious. Thus, in the majority of instances the referring physician tells the individual that he has pneumoconiosis or silicosis on the basis of a history of dust exposure, a physical examination which is thought to exclude other conditions that might produce similar symptoms, and the presence of abnormal shadows in roentgenograms of the chest which are compatible with those found in pneumoconiosis. The fact that diagnoses are made in this manner places a tremendous responsibility on the roentgenologist, for he must not only describe the roentgen appearances, but after he has done so, he must correlate other information which has been supplied to him with the roentgen observations, and arrive at a tentative or presumptive diagnosis. This is a safe procedure if we, as radiologists, fulfill our obligations to the referring physician and patient.

Criteria for Diagnosis[58]

The diagnosis of silicosis rests primarily upon a positive history of sufficient exposure to free silica dust plus the roentgenologic demonstration of characteristic deviations from the normal within the lungs. Physical and laboratory examinations are then required to exclude other conditions producing similar roentgenographic changes. Once the diagnosis is established, physical examination is required to determine the general physiological effect of the pulmonary condition and to determine whether any disability has resulted therefrom. Differences in individual incentive to work, the natural retardation of physiological responses with advancing age, and the accrued evidence to show that ordinary, slowly developing, simple silicosis is usually in itself nonproductive of a diminution in ventilatory capacity, all combine to make an accurate estimate of the latter an extremely difficult problem.

Machle,[44] however, has shown that the inhalation of the more active dusts results in certain changes in the behavior of the lung which favor retention of the very fine particles in larger numbers than would be expected to occur on a theoretical basis alone. The change is one of bronchiolar constriction, which leads to reduction in pulmonary volume and if continued leads to stasis with its attendant pathological changes. This is the type of observation which we hope to learn more about from pulmonary function studies.

Classification

Before discussing the roentgenographic appearances, it is necessary to say a few words about classification of silicosis as observed in the roentgenographic studies. There are many different ones in use. The most recent has been described by Fletcher[24] and his associates, who state that it may be necessary to have systems of classification for the various types of pneumoconiosis in men exposed to different dusts in various industrial processes. We have given the subject of classification or roentgen appearances considerable thought and feel that any classification used should not place too much emphasis on the roentgen observations, except in those instances in which there is good evidence of infection complicating the silicosis. A simple classification that has worked well for us for the last ten years is: *simple silicosis* and *silicosis with infection. It should be borne in mind that one cannot from a study of the chest by present roentgen methods give any reliable opinion as to the extent of disability.*

Simple Silicosis

The characteristic lesion in silicosis is a circumscribed nodule of hyaline fibrosis. The earliest lesions are invisible or are recognizable only microscopically or with a magnifying glass.[30] They are deposited along or within the lymph channels where they may impede lymphatic flow.[26,79] Gardner[32] states that when the lymph flow is retarded the phagocytes do not enter the lymphatics but collect here and there over the walls of the air sacs. Parenchymatous nodules may then develop which ultimately reach a size of 3 to 4 mm. in diameter. Pathologically, the nodules have well defined borders except when there are accumulations of non-siliceous dusts.

The earliest roentgen lesion that I accept as evidence of simple silicosis is the small, discrete, multiple shadow, 2-6 mm. in diameter, which is more or less uniform in size and density and does not disappear in a roentgenogram made with slight rotation. *Shadows that disappear with slight rotation are likely to be vascular.* The blood vessel shadows are denser and their borders are more sharply defined than are those of nodules. The shadows of the silicotic nodules are usually distributed along the vascular channels and the bronchial tree of both lungs, and at times they may be limited largely to one lobe. Not infrequently, even though a bilateral distribution of the

shadows occurs, one does not see them in the apical, peripheral and lower portions of the lung fields.

The roentgen appearance of the shadows of the silicotic nodules are not as characteristic as one would wish. Some are round, some oval, some irregular, some large and some small. Most of the lesions have a uniform density, but some have shadow densities in their centers simulating that produced by calcium. The shadows of these lesions are called nodulation, which unwittingly implies that they can be distinguished from mottling due to tuberculosis or some other infectious process. I frequently have found that the periphery of the nodular shadows (silicotic) is so ill defined that in itself it cannot be differentiated from mottling in miliary and other types of tuberculosis and small shadows due to a generalized metastatic malignancy. In most instances, however, a correct diagnosis can usually be arrived at by correlating the roentgen findings with the history and clinical data.

One of the interesting questions that arises concerning the silicotic individuals as seen by the roentgenologist is that not all of them develop generalized nodulation that can be seen on the roentgenogram. This apparently is true even when individuals are working under similar conditions in the same industry, and up to the present time, no adequate explanation has been made.

When nodulation occurs, the nodules may or may not show progressive changes. In a few patients who have been followed for fifteen years, we have observed the pattern of nodulation replaced by massive shadows with either complete or almost complete disappearance of visible nodular shadows on the roentgenogram. Just how often this occurs will be determined only after years of serial studies.

I have always been interested in whether nodulation is more likely to develop in the younger or the older individual. This question is likely to be answered as more experience with serial studies is obtained. My observations in one industry (almost pure silica) over a period of fifteen years tends to show that nodulation does occur more frequently in the young individual. These nodules have been observed in individuals who have had a silica dust exposure of five to ten years. One wonders whether the nodulation seen in some of the older individuals who have worked for longer periods may likewise have developed during the five to ten years' exposure period and in certain instances failed to progress.

Generalized nodulation is often present with very little, if any, clinical evidence of disability. Such an observation is extremely important to keep in mind for it will help to prevent such individuals from being thrown out of work and placed on compensation. Experience with such individuals has shown that if they are allowed to continue to work in a healthy atmosphere, the lesion may not progress, no unusual disability develops, and a family catastrophe or hardship due to loss of adequate compensation and unhappiness is prevented.

These nodules, uniformly distributed throughout the pulmonary parenchyma, form a characteristic shadow pattern, and *the demonstration upon the roentgenogram of this generalized nodulation is, from the radiologist's point of view, fundamental to the diagnosis.* There may or may not be associated enlargement of the hilar lymph nodes, despite the fact that it is in the lymphatic tissues that the earliest silicotic nodules have been shown to develop. These early nodules, however, by their very fibrous nature, are destined to cause eventual contraction, and in cases of slowly developing silicosis it may no longer be possible to demonstrate hilar lymph node enlargement by the time the parenchymal nodules are grossly visible.

Another lesion generally included under the classification of simple silicosis is the small conglomerate lesion. Such lesions may result from a combination or coalescence of discrete nodules or the lesion may occur as such primarily.

Conglomerate lesions are usually localized and do not occur in the same portion of the lung in every individual. On the other hand, roentgenographic studies of individuals with such lesions more frequently show them in the upper half of the lung fields. Microscopic examination[32] of the tissues from such areas oftentimes reveals no evidence of infection. The nodules seem to be closer together than in other portions of the lung; they are less uniform in size, and they are embedded in a matrix of diffuse, fibrous tissue having the same characteristic appearance as that forming the nodules themselves. It is possible that the conglomeration may have occurred because the portion of the lung in question was previously damaged by a localized, inflammatory process occurring before or during the early period of the dust exposure.[32] More dust would tend to ac-

cumulate in such an area and possibly the nodules would develop irregularly and closer together. Another explanation is that the scar contraction of the silicotic process may be sufficient to produce the conglomerate lesion.

The shadow of the conglomerate lesion in simple silicosis is often difficult to distinguish from that found in silicosis with infection. From the roentgenological standpoint they may be impossible to differentiate in a single examination, whereas serial examinations may or may not show slight changes in the extent of the shadows of lesions that are complicated by an active infection.

The roentgen observations in *simple silicosis* may be summarized as follows:

Trachea: Its shadow is in a normal position.

Heart and aorta: Their shadows are not noticeably affected by the silicotic process.

Domes of the diaphragm: The shadow and movement of the domes of the diaphragm are not affected by the silicotic process unless a coincident emphysema is present.

Hila: The shadows of these structures are as a rule not noticeably changed.

Trunk shadows and linear markings: Slight to moderate variations in these shadows are difficcult to evaluate.

Lung changes, nodulation and conglomerate lesions: The shadows of *nodulation* (2 to 6 mm. in size) are usually bilateral in distribution. Variations may occur such as a lobar or unilateral distribution.

Shadows of nodules may be difficult to differentiate from those occurring in some types of tuberculosis, metastatic malignancy and fungus infestations, et cetera.[54]

Conglomerate lesions: These shadows are usually found in the upper half of the lung fields and are difficult to evaluate in single examinations. Superimposed metastatic malignancy and lesions due to an active infection may produce similar appearances.

Hyperventilation and emphysema: Such conditions may be present and demonstrable by the roentgen examination. Their evaluation, as to cause and effect from dust exposure, however, is more difficult and does not fall within the province of the roentgenologist.

Silicosis With Infection

In this group are included all cases of silicosis with detectable evidence of infection. It is not always possible in the living patient to determine whether the infection is active or inactive even in instances when conventional clinical and laboratory examinations are available. Under such circumstances, mistakes will occur, but if one exercises good judgment, the affected individual can be protected by taking the necessary precautions.

The lesions (either some or all) that have been described as occurring in simple silicosis may be modified by infection. Other lesions that may be found include cavities (usually thick walled) tuberculous in origin, cavities occurring as a result of necrosis of anemic infarcts, massive lesions, mottling, soft nodulation, various degrees of emphysema and bleb formation, pleural thickening, pleural collections, pneumothoraces and deformations of the domes of the diaphragm. The roentgenologist is rarely able to predict whether the infection is due to the tubercle bacillus or some other organism. One suspects, however, that in the majority of instances, the super-imposed infection is tuberculosis, for Gardner[32] states that the postmortem examinations showed an element of tuberculosis in 60 per cent of the cases.

The changes occurring in the various structures of the respiratory and cardiovascular tracts as demonstrated by the roentgen examination may be summarized as follows:

Trachea.—Its shadow may be in a normal position, especially if a tuberculous process or some other infection is superimposed upon an already established silicotic process. In some instances the trachea is found displaced to one side. At such times, it is my feeling that the silicotic process has occurred either simultaneously with tuberculosis, or the tuberculous lesion was probably established before the silicotic changes became manifest, or the traction of the fibrotic changes produced by a tuberculous and silicotic lesion one side was greater than that of the silicotic process of the opposite side.

Heart and Aorta.—In some instances evidence of cor pulmonale is observed in the advanced silicotic individual. Some clinicians[17] have regarded this condition as a complication of silicosis. The relation between cause and effect is difficult to establish in this instance and cardiologists who

have given thought to the question find themselves unable to express an opinion. Many of us who have been interested in silicosis for years, have not been impressed with the incidence of cardiac complications. On the other hand, it is possible that with increased use of the electrocardiograph in the well advanced silicotic, more evidence of slight heart changes may be found. But even if such evidence is found, it is well known to the cardiologist and the general pathologist that in the older age group not exposed to harmful dust, which come to autopsy, cardiac complications are not uncommon. Even in the advanced silicotic with well established emphysema, Gardner[32] states that "hypertrophy of the right side of the heart without arteriosclerosis and even greater involvement of the opposite side is a rarity."

Occasionally, the heart and aorta are displaced to one side or backward by the contracting scar of a tuberculous infection and silicotic process. As in the case of the trachea and in the absence of data to the contrary, such displacement is thought to occur for the same reasons.

Domes of the Diaphragm.—If considerable emphysema is present, the domes of the diaphragm may be depressed and limited in their movements. At times it is necessary to have the patient cough in order to demonstrate, roentgenoscopically, evidence of diaphragmatic excursion.

Individualization of the costal components of the domes of the diaphragm is quite marked when there is considerable basal emphysema.

Another abnormal appearance is multiple peaking of the domes of the diaphragm. It may be impossible to differentiate the peaking caused by pleural adhesions from that due to inelasticity of certain structures of the lung. Both conditions may be present.

Hila.—The shadows of the hila may or may not be within normal limits. The hilum shadow may be enlarged or its shadow may be partly or totally obscured by a larger shadow produced by a lung lesion. The hilum may be displaced upward, laterally or backward by a contracting scar of a silicotic process plus an infection (similar to that causing displacement of the trachea).

Lung Changes.—Mottling is a term that is applied to the small and poorly defined shadows produced by an acute or chronic infection. The roentgen manifestations of mottling and nodulation may be identical and exceedingly difficult or impossible to differentiate. The shadows of mottling often lack a uniformity of distribution and are more likely to change in form in subsequent examinations than are the shadows of nodules. Mottling usually occurs as a result of a bronchogenic spread of a tuberculous lesion or the result of a fungus infestation.

Soft Nodulation.—The shadows of soft nodulation are much larger than those produced by nodulation and mottling, but are smaller than the conglomerate shadow. The description of such a roentgenologic shadow is provided in order to emphasize a perinodular cellular reaction that is observed by the pathologist. These shadows are much more likely to change in character in subsequent examinations than in the shadow produced by nodules, and change less than those due to mottling.

Nodulation.—The shadows of nodulation are similar to those described under "simple silicosis." Soft nodulation and nodulation cannot be demonstrated by a roentgen examination in every case of modified silicosis with infection. This is particularly true in certain industries such as hard coal mining and granite cutting. In cases where there is only roentgen evidence of massive or conglomerate shadows one is more likely to make errors in interpretation.

Massive Shadows.—These shadows vary from 3 to 20 cm. in size. Some are round, some oval and some wedge shaped. These lesions are usually due to extensive areas of fibrosis and are found in the upper third or upper two-thirds of the lung field. Occasionally it is possible with overexposed films, body section roentgenograms or a Potter-Bucky roentgenogram to show some of the details of a massive lesion, such as distorted and obliterated bronchi, cavities and areas of calcification and caseation.

A few years ago, most of these lesions were thought to be due to silicosis with infection. Riddell,[65] Gardner,[32] McCloskey[45] and others question whether this is always true. Gardner[32] thinks that a "third essential factor may be minerals other than free silica."

From a rontgenologic standpoint, I have attempted to determine whether the massive lesion

extended to the periphery of the involved lobe of the lung and have used this information as evidence for or against the presence of an active infection. For instance, if a lesion extends to the interlobar fissure or the thoracic wall, such a lesion would be regarded as the seat of an active infection. If the massive lesion were situated in the center of the affected lobe, the roentgen evidence would not necessarily be in favor of an active infection. I realize that such deductions by themselves are of very little value, but in the absence of more convincing clinical and pathological proof attention to this detail may prove to be helpful.

The massive lesions do not tend to displace the midline structures (trachea, hila, heart and aorta) by scar contraction. When displacements of those structures occur, the lesion which produces them is more likely to be either a tuberculous or Friedländer baccillus infection which has preceded or occurred simultaneously with the silicotic process. It should be possible however, for the contracting scar of a tuberculo-silicotic process of one lung to overcome that of an uncomplicated silicotic process on the opposite side.

Occasionally, one sees a lesion which by roentgen methods is diagnosed as a massive lesion, which at autopsy turns out to be a necrotic, anemic infarct or a cavity in which there is no microscopic evidence of infection and which is filled with a syrup-like material. I know of no way to diagnose the true nature of these lesions by the roentgen examination.

We have observed a migration of massive lesions toward the hilum in certain instances. This observation has been very helpful, and explains the prominent shadows adjacent in certain patients in whom there are large masses simulating greatly enlarged hilum lymphnodes.

The rationale for further division into the subgroups tuberculo-silicosis and silicosis with tuberculosis has already been explored, with emphasis placed upon the concept that tuberculo-silicosis is a separate and distinct, chronic disease entity resulting from the prolonged interaction of two disease processes, but differing radically in its behavior from either occurring alone.

The roentgen manifestations of tuberculo-silicosis are protean and yet distinctive. Early in its evolution there are characteristically present linear strands of fibrosis, presumably the result of previous infection, which however may be so fine as to be almost or completely obscured by the accompanying nodulation. In due course, serial roentgenograms reveal evidence first of concentration and later of coalescence of the nodulation about these foci of fibrosis. Newly developed areas of coalescence may extend to the pleural surface of the lungs, but if the disease remains chronic they inevitably contract to form the dense, well delineated "massive shadows" or areas of conglomerate fibrosis so typical of tuberculo-silicosis. These are located most often in the upper lung fields, but may radiate outward from the region of the hilum, occur as rounded masses deep within the lungs, or appear as wedges with their bases directed peripherally. Commonly, these areas of conglomerate fibrosis continue over the years to increase slowly in size, incorporating within themselves more and more of the individual silicotic nodules from other portions of the lungs, until finally one may have as the end-result either single or multiple, unilateral or bilateral massive shadows of conglomerate fibrosis. These may be so extensive as to destroy completely the ordinary identifying characteristics of both the tuberculosis and the silicosis, and they are invariably productive of an advanced degree of surrounding pulmonary emphysema. Since the affected individuals are not toxic, it is this latter which accounts for their obvious and often high degree of disability.

Only rarely is one sufficiently fortunate to see the evolution of the entire process in a given individual. It would seem however, that the pathologic evidence is sufficient, and that adequate numbers of cases have now been followed by means of serial roentgenograms over a period of years, to warrant a presumptive diagnosis of tuberculo-silicosis either upon the visualization of nodulation with concentration, coalescence or conglomeration, or, when bilateral, upon the demonstration by themselves of large areas of conglomerate fibrosis. Limited reservation must of necessity be entertained, however, until the case for or against non-tuberculous infection as the etiologic agent in the production of conglomerate fibrosis is definitely proved.

The behavior of many cases of tuberculo-silicosis would make it appear that the tuberculous component of the process is for a time held in check by the surrounding fibrosis. It remains a potential source of danger, however, and may become active at any time. Such activity manifests itself on the roentgenogram by the development of mottling and, once established, alters, by ac-

celeration of the fatal outcome, the entire course of the disease. It is still true, therefore, that a high percentage (though by no means all) of persons with tuberculo-silicosis ultimately die of tuberculosis.

Silicosis with tuberculosis, as previously indicated, may occur in either one or two forms, the clinical course and roentgen behavior of which are as distinctively different as is their pathology. Early in their development they may present an extremely difficult problem in diagnosis because of one's inability, except upon the basis of preceding serial roentgenograms, to formulate any estimate of the activity of the underlying silicotic process. In both groups the infectious component is first identified by the presence on the roentgenogram of single or multiple, usually apical, ill-defined areas of infiltration or mottling, superimposed upon a background of discrete nodulation. The subsequent behavior, however, is radically different.

In the first group—those in which we have fresh infection, either new or arising from the reactivation of a latent focus, superimposed upon a progressive and still active silicosis—the quartz is incompletely encapsulated by the still immature silicotic fibrosis and retains its ability to exert its specific effect. When the silica concentration is high, tiny foci of necrosis within the nodules occur, providing an ideal medium for the growth of tubercle bacilli; but even when the silica content is not excessive there is in some occult manner produced an environment eminently favorable to their multiplication.[28] Serial roentgenograms exhibit new areas of mottling and local extension of the original foci, about which the silicotic nodules may become extremely abundant.[29] In some, the so-called "perinodular type," there occurs, probably as a result of massive superinfection, a rapid enlargement with loss of definition of each nodule throughout both lungs, due to the development of an intense surrounding zone of collateral inflammatory reaction. Minute foci of caseation are usually present, and cavitation does occur, but by comparison with the general evolution of the process it is a relatively late phenomenon. While, rarely, several years may elapse before toxic symptoms supervene, the general tendency is toward uncontrollable extension to death before there is opportunity for the chronic changes of tuberculo-silicosis to occur.

In the second group are the unusual but existent cases in which silicosis and tuberculosis occur together but act independently. As previously indicated, it is presumed that in these cases the silicosis has healed with the formation of such densely fibrous nodules that the quartz is completely walled off and thus cannot exert its peculiar effect upon the superimposed tuberculous infection. The infection, observed by means of serial roentgenograms, is seen to behave typically as it does in non-silicotic subjects.

(To be concluded in the November issue.)

PAN-PACIFIC SURGICAL ASSOCIATION

The Fifth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, November 10-21, 1951.

The object of the Pan-Pacific Surgical Association is to bring together surgeons from countries bordering on the Pacific Ocean so as to permit the exchange of surgical ideas and methods and to develop a spirit of good fellowship among the various races represented. Although it was planned that meetings would be held every three years, world events have permitted but four conferences since the organization was conceived—the first in 1929 and the last in 1948.

The Fifth Congress provides an opportunity for doctors to combine a delightful vacation in Hawaii with attendance at a scientific meeting, the program of which will be presented by topflight surgeons from the Pacific area countries, as planned by the program committee. Doctors are urged to bring their families with them and are promised luxurious accommodations.

Dr. F. J. Pinkerton, president of the Association, has been officially appointed as travel agent for those coming to the meeting. To be assured of preferred accommodations, travel and hotel reservations should be made through Dr. Pinkerton.

Further information may be obtained by writing the Pan-Pacific Surgical Association office, Suite 7, Young Building, Honolulu, T. H.

THE PREDICTION AND PREVENTION OF CORONARY THROMBOSIS IN THE YOUNGER AGE GROUPS

A Suggestion for Further Study

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A NEIGHBORING doctor dies at thirty-seven from coronary heart disease; a farmer has a coronary infarction at thirty-four; a buttermaker has a coronary heart attack at forty-eight; the editor of a daily newspaper has a fatal attack at fifty-two. A World War veteran suffers an infarction at 53; another farmer, the same at fifty-two—all proven cases. These are by no means isolated instances but are selected at random from our small community within a short period of time.

We are not concerned here with coronary deaths among the aged or prematurely diseased. Those we may leave to their proper study in geriatrics. We are interested in the factors that produce coronary incapacity, causing such a high mortality in people in the fourth, fifth and sixth decades of life.

The purpose of this paper is to suggest a possible method of predicting coronary catastrophes and of possible prophylaxis for those people who are in their most productive years and who should be of most value to society.

The factors causing a coronary complex in the people in these earlier decades differ markedly from those in the aged. In older people, the primary cause of this trouble is an extensive intimal damage in the coronary arterial tree. Secondary factors are sedentary life, improper diet, and probably disease. In the younger years this initial damage is usually only moderate to minor. Several other factors apparently combine in varying importance to cause an infarction. Among these are prolonged nervous tension, the prothrombin time level, diet, and smoking. It is generally conceded that coronary constriction from chronic over-stimulation due to high nervous tension, worry, and overwork plays an important rôle in the foregoing disease entity. Therefore, we shall not add further comment. However, it is different with the other factors mentioned.

In the patients with coronary heart disease whom we have observed, we have been impressed by the low prothrombin time uniformly present.

TABLE I. PROTHROMBIN TIME BEFORE AND AFTER MIXED MEAL

Name	Approx. Age	Before Meal (Seconds)	After Meal (1 Hour) (Seconds)
G.P.	24	60*	55
A.G.	55	75†	60
S.L.	22	90†	75
B.J.	19	70*	65
A.R.	24	70†	60

*Light meal
†Moderate to heavy meal

TABLE II. PROTHROMBIN TIME BEFORE AND AFTER ALL-VEGETABLE MEAL

Name	Approx. Age	Before Meal (Seconds)	After Meal (Seconds)
B.J.	19	55	45
A.G.	55	45	45
S.L.	24	45	40
L.K.	22	50	45
A.R.	24	45	50

TABLE III. PROTHROMBIN TIME BEFORE AND AFTER SMOKING

Name	Approx. Age	Fasting 7 a.m. No Liquids (Seconds)	Fasting ½ Hour After smoking 2 Cigarettes (Seconds)
B.J.	19	60	45
C.T.	40	70	60
J.C.	40	45	35
G.A.	45	65	50
A.R.	24	60	45

Likewise, we have been impressed by the fairly prompt relief of pain in the non-fatal cases when the prothrombin time has been quickly elevated by the use of the anti-coagulants, heparin and dicumarol. We also noted that the pain did not often recur if the prothrombin time level was sustained at a sufficient height. Pain, however, was most likely to recur following a meal. Knowing of no explanation for this, we ran a short series of prothrombin time levels (Smith bedside whole blood method) on normal individuals immediately before eating and again an hour afterwards. The results are shown in Table I.

As a variation, we also ran a series before and after an all-vegetable meal (Table II).

These tests suggest diet as an important factor in precipitating the coronary syndrome.

(Continued on Page 1003)

THE HEART IN FRIEDREICH'S ATAXIA

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FATTY infiltration of the heart was noted by Friedreich in the original description of the disease that still bears his name. This was in 1863. The hearts in which he noted these changes, however, were those of patients who had died of typhoid fever, and there is no way of knowing whether the changes which were noted were due to Freidreich's ataxia or to typhoid fever. The occurrence of cardiac failure as a terminal event in this disease was emphasized by Pitt as early as 1886. From this time on, scant reference[8,9] was made to the cardiac manifestations of Friedreich's ataxia until the report by Loiseau in 1938. He reviewed the literature and found reports of 40 cases in which there were cardiac abnormalities. It was his belief that these cardiac abnormalities were incidental in some cases and specific to the disease in others.

In 1942, Evans and Wright reviewed the literature and reported on the electrocardiographic findings in Friedreich's ataxia. Russell in 1946 gave a detailed and accurate account of the pathologic changes found in the hearts of persons affected with this disease. Since then, scattered reports[5,10,17] have further emphasized the involvement of the heart in Friedreich's ataxia. From the evidence that is available, it is clear that certain cardiac abnormalities are definitely related to Friedreich's ataxia. These do not include such disorders, either acquired or congenital, which exist coincidentally, or the myocarditis secondary to intercurrent viral or bacterial infection which may occur in persons who have Freidreich's ataxia.

The usual clinical manifestations indicative of myocardial involvement are disturbances of rhythm and myocardial insufficiency terminating in congestive heart failure. Complete heart block with Stokes-Adams syndrome has been observed by Evans and Wright in siblings aged eighteen and twenty-two years. Piron believed angina pectoris was present in two brothers he observed.

Examination of the heart may reveal any of the cardiac arrhythmias alone or in various combinations. The heart may or may not be enlarged, and, in those patients who have acquired a kyphoscoliosis, it is displaced. Systolic and diastolic murmurs have been observed infrequently. Hejtmancik, Bradfield and Miller emphasized the diastolic murmurs heard in their two cases and in several cases reported in the literature, but murmurs of any type, especially diastolic, are the exception rather than the rule and are due to cardiac dilatation rather than to valvular disease.

The roentgenologic appearance of the heart is variable. The heart may be of normal contour or it may be globular; it may show varying degrees of left ventricular enlargement or generalized dilatation. In general, cardiac enlargement or dilatation is seen only late in the disease, and in association with far-advanced neurologic disease.

The electrocardiographic abnormalities are of two distinct groups:[11,15] those showing disturbances in impulse initiation and transmission, and those showing alteration of the ventricular complex. The former group includes such arrhythmias as paroxysmal auricular tachycardia, ventricular tachycardia, auricular fibrillation, auricular flutter, varying degrees of auriculoventricular block including first degree, second degree and complete heart block and the Wenckebach phenomenon, right bundle branch block, and various combinations of these disorders. The recorded observations are not sufficient to permit one to make a statistical analysis, but paroxysmal auricular tachycardia and auricular fibrillation are more commonly encountered than any other arrhythmia.

The modifications of the ventricular complex include slurring or low voltage of the QRS complex, deep Q waves, elevation or depression of the ST segment, and sharp, pointed, iso-electric diphasic or inverted T waves in one or more leads. These abnormalities may occur alone or in any combination. Great emphasis has been placed on the occasional finding of a Q_1 T_1 or Q_3 T_3 pattern simulating that found with anterior and posterior myocardial infarction, respectively, and on T_1 or T_3 patterns with "coronary type" T waves. However, when studies with multiple precordial leads were made,[6,17] evidence of a local myocardial lesion was always lacking.

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The study of Evans and Wright of 38 patients with Friedreich's ataxia is significant in that they included patients whose disease from the neurologic standpoint was less far advanced than that of patients reported by other authors. They found definite abnormalities in the electrocardiogram of twelve of these patients, and, in all but one, who had complete heart block, the abnormalities were confined to the QRS complex. The changes were chiefly inversion of the T wave in one or more leads. Occasionally, all the limb leads showed these changes, and, in addition, some abnormalities occurred in widely separated precordial leads. They found that absent tendon reflexes and extensor responses of the plantar reflex, indicating more advanced neurologic disease, were more common in patients with abnormal electrocardiograms. Generally speaking, neurologic signs were more widespread in the patients who had the most conspicuous electrocardiographic changes. Electrocardiographic abnormalities also occurred more frequently in cases in which there was a family history of Friedreich's ataxia, and, finally, there was a marked tendency for members of the same family to have similar electrocardiographic patterns.

The pathologic changes considered by Russell to be characteristic although not pathognomonic consist of patchy loss of muscle fibers, infiltration with round cells and leukocytes, hypertrophy of muscle fibers, and separation of these fibers by an increase in fibrous or collagenous interstitial tissue. Pericarditis, endocarditis or organic valvular deformity was not noted by her. The presence of an interstitial myocarditis had been mentioned in a few reports before[12,13,16,18] and since[5,10] this study by Russell. She expressed the opinion that a focal, piecemeal coagulation necrosis of the muscle fibers takes place and is followed by cellular infiltration. As a result, the fibers are ultimately replaced by collagenous tissue, and the surviving muscle fibers undergo compensatory hypertrophy. This continues over a long period of time, until heart failure develops. At this terminal stage, a severe fatty degeneration is usual.

The theories advanced concerning the etiology and pathogenesis of the myocarditis and arrhythmias include the bulbar, the coronary artery, the infectious and the toxic theories.[11,15]

The bulbar theory, favored by the French,[1,2,8,9,19] is based on the occasional association of injury of the vagal nuclei and evidence of imbalance of the autonomic nervous system, such as paroxysmal cardiac arrhythmias, Cheyne-Stokes respiration, episodes of acute abdominal pain with ileus, and disturbances in temperature control, urine formation and sweating mechanism. However, these clinical findings are not present in the majority of cases, and Russell was unable to find evidence of injury of the vagal nuclei in her cases.

The coronary artery theory is based on the superficial resemblance of the described electrocardiographic changes to the patterns seen in myocardial infarction.[4] However, complete electrocardiographic studies with multiple precordial electrodes show no evidence of the focal type of myocardial injury[6,17] seen in myocardial infarction. Coronary artery disease was not evident at necropsy,[5,10,12,13,16,18,20] and injection of the coronary vessels showed no obstruction in even the finer ramifications of the coronary vessels.[14]

While it is impossible to prove that the myocarditis is not the result of either old or a recent infection so mild as to escape notice, its high incidence in patients with Friedreich's ataxia and its complete absence in unaffected members of the same family suggest that this is not the case. The progressive nature of the myocarditis over a period of several years is more suggestive of the continued action of some unknown agent than it is of the single insult of an acute infection. On the basis of pathologic findings, Russell, Lambrior, and Lannois and Porot concluded that the myocarditis is a result of toxins instead of infection. Available evidence suggests that the most valid theory is that the myocarditis is due to the action of an unknown toxin on heart muscle which may be abnormally susceptible to that toxin because of hereditary influences. It cannot be denied that purely neurogenic influences could be a factor in the production of the arrhythmias[3] but it is not necessary to postulate this mechanism in view of the presence of diffuse morphologic changes in the myocardium.

Contrary to current teachings that patients with Friedreich's ataxia die of intercurrent infection, it would seem that death due to an associated myocarditis is not infrequent. The appearance in these patients of any cardiac abnormality, either clinical, radiographic or electrocardiographic, is probably indicative of a poor prognosis. Certainly, cardiac enlargement, a major arrhythmia or congestive heart failure usually means

death within a year or two. The course of the disease moreover does not seem to be influenced by the usual measures which are ordinarily effective in the control of congestive heart failure.

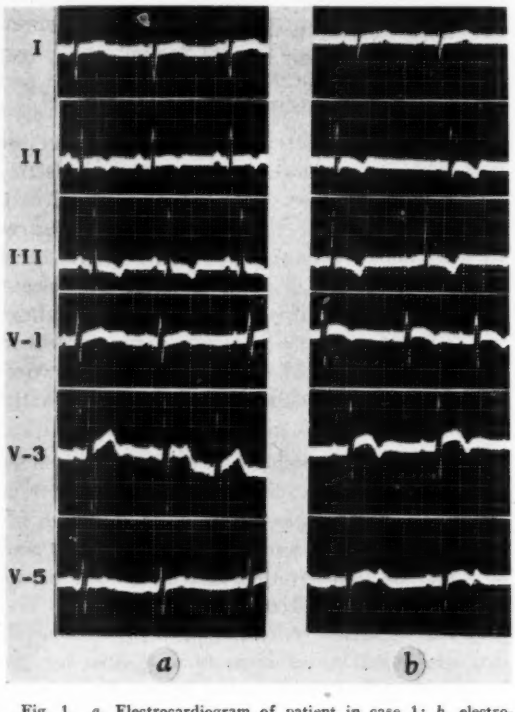

Fig. 1. *a*, Electrocardiogram of patient in case 1; *b*, electrocardiogram of patient in case 2.

Despite this a trial of therapy including digitalization, diuretics and quinidine in selected cases seems worth while.

Report of Cases

Case 1.—An eighteen-year-old white man, who was a high school graduate, was brought to the Mayo Clinic on August 4, 1949, because of difficulty in walking. He had been delivered with the aid of forceps but his neonatal and infancy development had been normal. At the age of six years, it had been noted that he was unable to run, jump or hop and that he had difficulty in walking because of unsteadiness and inco-ordination of the ankles. Soon thereafter, slight inco-ordination of the upper extremities had been noted. These difficulties had progressed gradually without remission during the subsequent twelve years. His general health had been excellent. He had not had any cardiorespiratory symptoms, or indeed any symptoms except those referable to the neurologic disorder.

Physical examination revealed an adequately developed and nourished white male of 18 years with moderate kyphoscoliosis but no deformity of the feet. The cardiorespiratory system was completely normal as were the results of the remainder of the general examination. Neurologic examination revealed a marked ataxia, absence of all tendon reflexes, and bilateral extensor responses of the plantar reflex. It also revealed gross inco-ordination with dysmetria and adiadokocinesis. Romberg's sign was present. The joint sense in the great toe was absent, and there was delayed pain sense in the feet. Nystagmus was absent. The ocular fundi were normal.

The results of routine laboratory tests and roentgenographic examination of the heart, lungs, head and pelvis were normal except for the presence of kyphoscoliosis. The electrocardiogram (Fig. 1*a*) was abnormal, for it showed right axis deviation, occasional auricular extrasystoles, notched QRS_1, slurred QRS_3, inverted T_2 and T_3 and diphasic T in lead V_5. The T wave was positive in leads V_1 and V_3.

A diagnosis of Friedreich's ataxia with myocarditis was made.

Case 2.—The twelve-year-old brother of the patient in Case 1 was also seen at the same time. He had nearly identical but milder symptoms. The onset of his disease was uncertain but it probably had occurred when he was between 6 and 8 years of age. It then had been noticed that he was awkward in running and walking and often stumbled or fell. There had been little or no progression of his symptoms since they first had been noted. As with the brother, there was complete absence of cardiorespiratory and other symptoms.

The results of general physical examination were normal except for the presence of slight lumbar scoliosis. Neurologic examination revealed less marked ataxia and inco-ordination, but the same reflex changes as were present in the older brother, namely, absent tendon reflexes and bilateral extensor plantar reflexes. Sensation, however, was normal. There were slight nystagmoid motions but definite nystagmus was not present.

The results of routine laboratory tests and roentgenographic examination of the heart, lungs, head and spinal column were normal except for the presence of slight lumbar scoliosis. The electrocardiogram (Fig. 1*b*) was even more abnormal than that of his brother. It showed right axis deviation, slurred QRS_1, inverted P_3, inverted T_2 and T_3, inverted T and elevation of the ST segment in leads V_1 and V_3 and diphasic T in lead V_5.

The diagnosis in this case also was Friedreich's ataxis with myocarditis.

The parents and two remaining siblings, aged thirteen and sixteen years, were then examined. None had symptoms of neurologic or cardiorespiratory disease. There was no evidence of Friedreich's ataxia, pes cavus or cardiac disease. Their electrocardiograms were all normal.

Comment

Our observations on these two brothers are partly in agreement with those of Evans and Wright in that the two neurologic signs most frequently seen in patients with electrocardiographic

abnormalities, namely, an absence of tendon reflexes and an extensor response of the plantar reflex, were present in both cases.* They represent another example of the observation that afflicted members of one family tend to have similar electrocardiographic patterns. We have no explanation for the finding of more marked electrocardiographic changes in the younger of the two patients in whom neurologic damage was less marked, but emphasize it because it is contrary to previous experience. The extension of electrocardiographic abnormalities across the entire precordium in this latter case gives further support to the belief that most, if not all, of the cardiac abnormalities associated with Friedreich's ataxia are due to a diffuse myocarditis. The cause of this myocarditis is unknown but it is probably of toxic cause.

*Since this paper was completed, the report of G. W. Manning (Cardiac Manifestations in Friedreich's Ataxia. Am. Heart J., 39:799-816 [June] 1950) has been published. It emphasizes the clinical and electrocardiographic aspects of the cardiac arrhythmias and heart failure noted in four of six patients with Friedreich's ataxia. Myocarditis similar to that described by Russell was found at necropsy in one case.

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CORONARY THROMBOSIS IN THE YOUNGER AGE GROUPS

(Continued from Page 999)

This further observation was made. If the patient was allowed to smoke cigarettes, he frequently was threatened with a syndromal attack. On suspicion, these few tests were run on normal people (Table III).

From this meager evidence, it may be that cigarette-smoking acts not only to constrict the arterial lumen but, what may be more dangerous, to lower the prothrombin time level.

It is known, of course, that a high percentage of people subjected to these same conditions, tension, dietary indiscretions, and smoking, survive into an older age group. There must, then, be some common factor that determines the precipitation of coronary attacks at different ages. We believe this factor to be the difference in prothrombin time level. We believe that this difference in level is a hereditary factor, and that it plays an important rôle in coronary attacks. Therefore, we believe that coronary attacks are predictable and preventable.

Assuming, then, that this reasoning is correct, should it not be reasonable to assume that by routine testing, individuals prone to coronary disease could be detected and prophylactically treated with dicumarol to elevate the prothrombin time to a satisfactory and safe level just as the diabetic individual can be detected and treated?

We realize, of course, that a prolonged research would be required to establish the proof of this reasoning. Even a few hundred selected people tested and followed through the years should establish the veracity of this contention. We believe, however, that such research in prothrombin time levels would be justified in an attempt to prevent the colossal cost in heart deaths exacted by our modern high-geared society.

CHRONIC LEUKEMIC INFILTRATION OF THE GASTRIC WALL SIMULATING PEPTIC ULCER

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THE FIRST description of gastric involvement in a case of apparent leukemia was given by Briquet in 1838 and published in Cruveilhier's Atlas of Anatomy. In this case, the mucosa of the stomach and bowel was thrown into folds resembling cerebral convolutions. Since then, as reported by Pearson, Stasney and Pizzolato,[11] in all descriptions of autopsies on lymphatic leukemia patients, various authors have stressed the diffuse involvement of the mucosa and submucosa of the stomach, usually without involvement of the deeper layers. These authors reported two similar cases.

Various pathologists since Cohnheim have recognized anatomical changes in various organs, in leukemia, without changes in the blood. Cohnheim[2] suggested the name "pseudoleukemia." Warthin[15] designated the condition a generalized or localized "aleukemic lymphocytoma." Ewing[3] observed: "The gastrointestinal tract is a seat of a remarkable form of primary lymphoid hyperplasia which lacks the destructive character of lymphosarcoma and fails to give lymphocytosis of the blo[illegible] The process may be chiefly limited to a portion of, or involve the whole of, the gastro-intestinal tract, or it may be associated with widespread lesions of most of the other lymphoid structures." Ewing[3] designated this as "aleukemic lymphomatosis." Ikeda[5] in 1931 described a case of gastric tumor and infiltration thought to be carcinoma, on whom a gastro-enterostomy was done. After five years the patient died of almost generalized lymphatic leukemia and heart failure. The entire gastro-intestinal tract was involved. In this case the infiltration extended out into the muscularis and serosa. The mucosa in the upper part of the stomach showed the thickened rugae described by Briquet. Ikeda[5] found, in 12,396 autopsies at the University of Minnesota, seventy-seven cases of leukemia of which fifty-one were of the lymphatic type and twenty-six of the myelogenous type. Of the former, only two cases showed local nodular elevation or thickening of the gastric wall, and two showed ulcers. In the myelogenous type, only one showed a local thickening of the mucosa. Grossly these lesions could not be differentiated from those of Hodgkin's disease or of lymphosarcoma. Ikeda[5] recommended abandoning use of the term "pseudoleukemia gastro-intestinalis" since it included all of the above types of lymphoblastomata. According to Mead,[8] the pathologic character of lymphatic leukemia of the gastro-intestinal tract varies from slight swelling of the mucous membrane and lymph follicles to extensive hyperplasia of the lymphoid tissue of the entire gastro-intestinal tract, with associated generalized lympadenopathy. The process may be limited to one organ or to the whole gastro-intestinal tract. Polypoid formations anywhere in this tract are common. The stomach mucosa often exhibits enlarged convolated rugae. Ulceration of the mucosa is infrequent. Mead[8] states that the muscularis is uniformly uninvolved. Gastro-intestinal Hodgkins Disease is uncommon but ulceration is relatively common in this disease as it is in lymphosarcoma. In the leukemias, as recorded by Paul and Hendricks,[10] one often finds small areas of thickening and infiltration of the mucosa, and in such thickened areas the mucosa may become denuded resulting in shallow ulceration and hemorrhage.

Forkner[4] states that pseudoleukemia (aleukemia) seems more likely to give gastro-intestinal involvement than do cases where the blood picture is positive for leukemia. He also emphasized the difficulty of differentiating the gross alterations in the gastro-intestinal tract caused by leukemia, aleukemia, lympho-sarcoma and Hodgkin's Disease. In contrast to the infrequentcy of leukemia of the gastro-intestinal tract, O'Donohue and Jacobs[9] collected a series of 100 cases of

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lymphosarcoma of the stomach reported by various authors from 1937 through 1946. Poer[12], Macchi[7] and Koucky, Beck and Atlas[6] have given separate descriptions of acute perforations of lymphosarcomatous ulcers of the stomach and duodenum which presented clinically as peptic ulcers. This is extremely rare in leukemia.

The intensity of symptoms in lymphatic leukemia of the gastro-intestinal tract seems to have no constant relationship to the extent of the pathologic changes. The most extensive lesions may be symptomless. Achlorhydria is an occasional finding. Areas of hemorrhage or small ulcers due to secondary infection may occur in some cases. There may be profuse hematemesis, bloody diarrhea, loss of appetite, weight loss and weakness. An abdominal mass may be present. These symptoms cannot be differentiated from those of other lymphoblastomata or of carcinoma. The x-ray is of little value in diagnosing any lymphoblastoma; most cases have been called carcinoma. Ikeda[5] states that the demonstration of deep heavy rugal impressions on the x-ray, together with a postive blood picture or biopsy, may be helpful in diagnosis of gastric leukemia but he emphasized that there is no pathognomonic roentgen picture of the stomach in the gastric manifestation of lymphatic leukemia.

The use of the gastroscope in the diagnosis of lymphoblastoma of the stomach was introduced by Schindler[14] in 1922. However, he stated in his first edition in 1937 that no case of leukemia or aleukemia of the stomach had ever been observed gastroscopically. In his 1950 edition he states that gastroscopy often yields important results in leukemia. Hypertrophic gastritis may be seen in myeloid leukemia and atrophic gastritis is more apt to be seen in lymphatic leukemia. Leukemic infiltrations are visible at times. Schindler[14] finds that the differentiation between Hodgkin's disease, lymphosarcoma, leukemia, some types of gastritis, carcinoma or syphilis of the stomach cannot be made from the gastroscopic picture alone. The diagnosis can be made only if biopsy of a lymph node reveals the typical picture and a diffuse stiff infiltration is seen. Leukemic infiltration and Hodgkin's disease of the stomach will be suspected only if other signs of these conditions are present elsewhere. In difficult cases, Schindler[14] recommends biopsy at laparotomy without the intent of resection. The biopsy should be taken from the area which was suspicious at gastroscopy. The abdomen then is closed and paraffin microscopic studies of the biopsied tissue are run. In lymphoblastoma, irradiation then is used. If syphilis is found, treatment is started. Carcinoma is promptly resected.

Renshaw and Spencer,[13] in 1947, found no reports available of the gastroscopic appearance of leukemic, pseudoleukemic or Hodgkin's infiltrations of the stomach, but judging from autopsy descriptions of such lesions, these authors were of the opinion that the lesions would be indistinguishable from carcinoma or lymphosarcoma. They described the gastroscopic appearance of eight cases of gastric lymphosarcoma, in six of which a diagnosis of infiltrating or ulcerative carcinoma was made. In the other two cases, cervical lymph nodes pointed to the proper diagnosis. They state that there is no characteristic gastroscopic picture of lymphoblastoma.

The gastroscopic examination of patients with malignant lymphomata at the University of Iowa has been well described by Paul and Hendricks.[10] All patients at that institution who are found to have any type of lymphoma are routinely gastroscoped. From 1941-1947, 53 patients with malignant lymphoma were found by such examination to show characteristic involvement of the stomach while during this same period only fifteen cases were found at autopsy to have gastric lymphomatous lesions. Of these fifty-three patients, twenty-two had chronic lymphatic leukemia, ten had myelogenous leukemia, one had monocytic leukemia, and twenty had Hodgkin's disease. There was no correlation in these cases between the type and duration of symptoms, degrees of free HCl or the extent of involvement. The ages ranged from nineteen to seventy-five years. The authors state that the gastroscopic picture was no indication of the type of hematopoietic neoplasm present. The most frequent gastroscopic finding was a granular mucosa suggestive of "goose pimples," seen usually on the anterior wall near the angulus (Depth II). The next most common site was the posterior wall of the antrum. These granular elevations appear as highlights on a dry mucosa and at times are 1 mm. in diameter and large enough to project above the surface. The same appearance was found in some of these patients in the mucosa of the rectum and sigmoid. Here they differ from the mucosal findings in chronic ulcerative colitis in being fewer in number and of larger size. The color

of the gastric mucosa in these cases varied with the degree of anemia present and the mucosa often presented gelatinous areas of edema with small hemorrhages. The second type of lesion

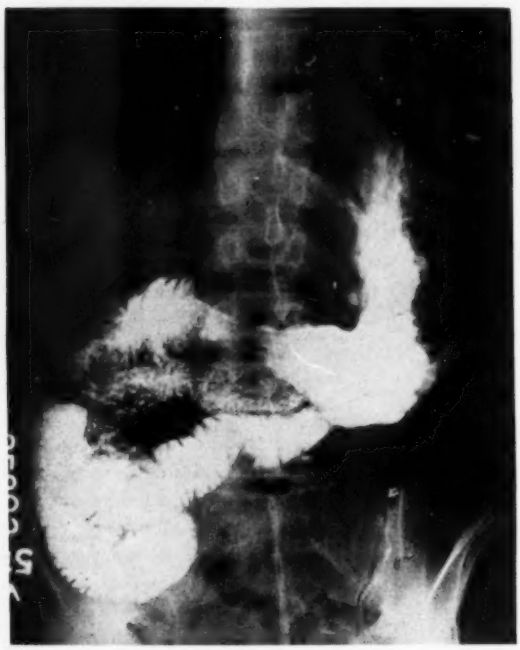

Fig. 1. Large penetrating gastric ulcer on the lesser curvature just above the incisura. It appeared benign both to the roentgenologist and to the gastroscopist.

seen through the gastroscope in these lymphoma cases was a nodular mass, not over 1 cm. in diameter, surrounded by abnormal appearing mucosa, often hemorrhagic. These usually were near the cardia, more often on the posterior wall. One such nodule was later examined at necropsy and found to be an infiltration of lymphatic leukemia. The third, and least common type of lesion seen in these cases was an ulceration. Two such ulcers were found, both in Hodgkin's disease, and both were on the greater curvature. The authors conclude that hematopoietic neoplasms involve the stomath with greater frequency than the medical literature would indicate. Since the mucosa and submucosa are involved, gastroscopic evaluation is made possible and is very helpful.

Very recently, Benedict[1] has reported on the use of the flexible operating gastroscope in the diagnosis of lesions of the stomach. He reported such biopsies to be of definite value in the diagnosis of lymphoma, carcinoma and gastritis. Sixty-three biopsies had been done without accident or complications. For obvious reasons, the method has not been used or recommended for differentiation of benign from malignant gastric ulcers. Since lymphoma is usually a diffuse process, Benedict[1] thinks that a negative biopsy probably excludes this diagnosis with reasonable certainty. (In view of the work just described, by Paul and Hendricks,[10] such an assumption might be open to question.) If doubt exists and lymphoma seems likely clinically, gastroscopy and biopsy should be repeated, taking the latter from a new area of the gastric mucosa.

As to treatment of the gastric lesions of hematopoietic neoplasm, Paul and Hendricks[10] report that irradiation and nitrogen mustard had little, if any, effect on the gastric involvement. In some cases the lesions progressed after these agents were used. Watkins,[16] however, has found considerable benefit from the use of nitrogen mustard in adequate dosage. Schindler[14] recommends surgical biopsy and if this is positive for lymphoma, irradiation is then given. Mead[8] stated that the best results in treating lymphatic leukemia of the stomach were obtained by combined surgery and x-ray therapy.

Case Report

A white, retired postal employe, aged fifty-nine, was admitted to the Minneapolis Veterans Hospital on October 7, 1949. The patient was complaining of generalized malaise, sore throat of two weeks' duration, rhinitis and sinusitis of three weeks' duration and inability to maintain himself on his ulcer regime at home.

Past history revealed that his first admission to the hospital was in 1931 at which time a diagnosis of chronic duodenal ulcer was made; the diagnosis was confirmed by roentgenologic study. In 1940, he was readmitted because of persistent vomiting, epigastric pain and a weight loss of 25 pounds. A duodenal ulcer was again demonstrated and the symptoms promptly subsided with conservative therapy. Precordial pain and shortness of breath were listed as additional complaints.

A severe attack of precordial pain, with radiation of pain into the left arm and hand, associated dyspnea and profuse diaphoresis necessitated emergency hospitalization in 1945. Therapy was exercised in keeping with a diagnosis of acute coronary occlusion. However, serial electrocardiograms failed to reveal infarction, or evidence for coronary insufficiency. Numerous electrocardiographic studies done since that date have continued to demonstrate normal tracings.

Precordial pain of ten days' duration, partially relieved by sublingual nitroglycerine, was the inciting cause of his readmission in June, 1947. Laboratory studies at this time revealed a persistently elevated white blood cell count ranging between 17,000 and 32,000. Biopsy of a small node in the right axilla revealed

chronic lymphatic leukemia; this was confirmed by bone marrow biopsy. X-ray films of the upper gastro-intestinal tract were again made, which revealed a filling defect in the midportion of the body of the stomach. This was thought to be of questionable significance; duodenal deformity was again noted. From June, 1947, until January, 1949, follow-up studies at three-month intervals were carried out. At the time of each reexamination, precordial pain and epigastric distress continued to be the main complaint. During this interval the spleen and liver became palpable and the lymph nodes became more prominent. Repeated white blood cell counts were in the range between 60,000 to 80,000 with 80 to 90 per cent mature lymphocytes; occasional immature lymphocytes were observed.

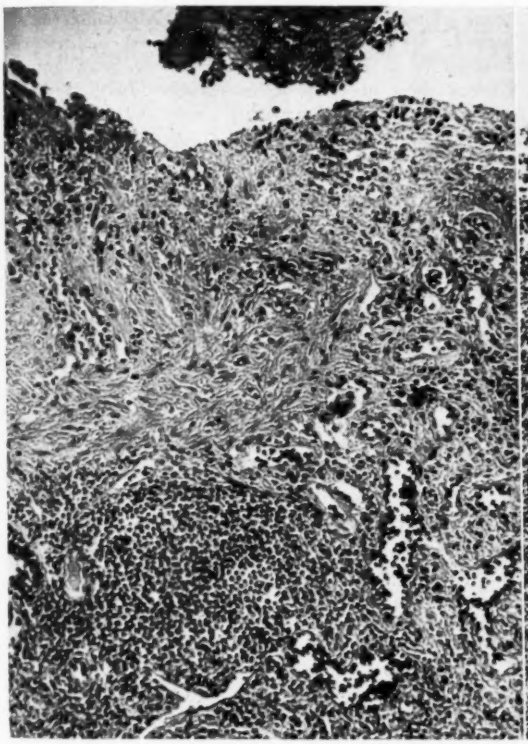

Fig. 2. Microscopic section through the base of the gastric ulcer. Necrotic debris, proliferating fibroblasts, and an underlying diffuse lymphocytic infiltration are noted.

Fig. 3. One of the large collections of lymphocytes found in the submucosa. Others were found in the muscular layer and in the serosa.

During the latter part of January, 1949, the patient was again admitted as an emergency because of nausea, vomiting, 10 pound weight loss, epigastric distress, and precordial pain. An upper gastro-intestinal study was done and a gastric ulcer was observed on the lesser curvature and posterior wall (Fig. 1). A crater in the duodenal bulb was also noted. Gastroscopy was carried out and a large penetrating gastric ulcer on the lesser curvature about one inch above the incisura was observed. The crater was approximately 1.75 cm. in diameter. The membrane lining the crater had a smooth greyish white color. The rim of the crater was moderately edematous and only slight injection surrounded the rim of edema. Gastroscopy and x-ray studies were carried out at two-week intervals. Examinations done seven weeks after the first examination revealed complete healing of the previously reported ulcer.

Despite visits at two months' intervals, the patient sought emergency readmission in August, 1949, because of intractable epigastric distress, nausea, vomiting and a 15-pound weight loss, which had occurred in a period of four days. Fluid balance was restored and ulcer management was continued. Nightly aspirations were carried out and residual retention ranged from 50 c.c. to 300 c.c. The patient was taught to use an Ewald tube. It was decided that with lymphatic leukemia, and coronary insufficiency, surgery should be deferred until it was actually imperative to relieve obstruction. Gastro-intestinal studies at this time demonstrated a minimal residual of the gastric ulcer on the lesser curvature and a duodenal deformity.

At the time of admission on October 7, 1949, physical examination revealed a thin, undernourished male who appeared acutely ill. The blood pressure was 142/76, pulse 116, respirations 22, temperature 101.4° Fahrenheit. The mucous membrane of the nose and throat were congested and a purulent post-nasal drip was observed. Signs of right upper lobe consolidation were present. A soft systolic murmur was heard at the apex and the

heart was enlarged to the left. The liver and spleen were palpable on deep inspiration. Tenderness and muscle guarding were present in the midepigastric region. Shotty nodes were found in the cervical chains bilaterally and both axillary and inguinal regions.

The hemoglobin was 12.1 grams, white blood cell count 83,000 with 15 per cent neutrophils and 85 per cent mature lymphocytes, sedimentation rate 98 mm. per hour, serum proteins 6.7 grams with 4.4 grams of albumen and 2.3 grams of globulin. Prothrombin time was 100 per cent of normal. Bromsulfalein: 2 per cent retention in 45 minutes. Serum bilirubin: 1 minute, 0.2 mg. per cent; total, 0.5 mg. per cent. Gastric analysis: total acid 44°, free acid 36°. Stools were negative for ova, parasites and occult blood. Urinalysis showed a trace of albumen and an occasional white blood cell. Sputum cultures revealed the usual mouth organisms. X-ray studies of the chest revealed a diffuse infiltration in the right upper lobe. Electrocardiogram again showed a normal tracing.

The patient was placed on penicillin and the fever promptly subsided; however, pleural effusion developed and required repeated aspirations. Cultures of the aspirated fluid were repeated negative. Following subsidence of the inflammatory process an upper gastrointestinal study was carried out and an ulcer crater was visualized on the lesser curvature. Gastroscopic study at this time demonstrated a one cm. crater, penetrating in nature, 2 cm. above the incisura slightly to the posterior wall side on the lesser curvature. The ulcer crater was covered by a smooth glistening gray membrane. No heaping up of the ulcer margin was seen; a slight rim of edema surrounded the crater.

In spite of intensive therapy the crater continued to enlarge and the patient was transferred to the surgical service. Two days prior to scheduled surgery, the patient became faint, weak and passed numerous tarry stools. The hemoglobin level dropped to 7.9 grams. Three thousand c.c. of whole blood were required to restore his hemoglobin level to 14 grams. Subtotal gastrectomy was carried out and the postoperative course was uneventful.

Examination of the resected portion of the stomach revealed a depressed area on the lesser curvature which appeared to be a healed ulcer. Immediately adjacent to this area, an elliptical ulcer with slightly rolled edges 1.2 by 2.5 cm. was present. The base of the ulcer was necrotic but was not indurated. In the pyloric portion of the stomach, on the anterior wall and midway between the greater and lesser curvature, an old healed ulcer was represented by puckering at this spot.

Microscopic studies revealed the base of the ulcer to be composed of necrotic debris and some slight proliferating fibroblastic tissue (Fig. 2). The ulcer was relatively superficial, extending only into the inner muscular coat. The mucosa became thinner and just faded off at the edge of the ulcer. Underlying the ulcer was a diffuse lymphocytic infiltration which extended through the musculature and became particularly heavy in the serosa. Large collections of lymphocytes were found in the submucosa, musculature and serosa of the stomach wall near the ulcerated area, (Fig. 3) and similar collections were found in all portions of the stomach sectioned, whether near or far from the area of ulceration. These masses of lymphocytes were found diffusely throughout the stomach wall and were arranged in such discrete units that they did not give the appearance of an inflammatory process, but rather of leukemic infiltration of the stomach wall. Abdominal nodes removed at the time of surgery showed the normal architecture to be completely wiped out by a homogeneous sheet of lymphocytes consistent with a diagnosis of lymphatic leukemia.

Conclusions

1. The literature is reported describing the gastric manifestations of lymphatic leukemia.

2. Ulceration of the gastric mucosa is extremely rare in lymphatic leukemia of the stomach. When present it is almost always of a superficial type, amounting only to small erosions. Deeper ulcers, resembling peptic ulcers in a nonleukemic stomach, have not been described in the literature.

3. A case is reported where recurrent ulceration, resembling peptic ulceration, both by x-ray and gastroscopy, was found on the lesser curvature of the stomach in a patient with proved systemic lymphatic leukemia. Gastric resection was finally performed. The pathological examination of the resected specimen revealed typical chronic lymphatic leukemia infiltration of all layers of the gastric wall.

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CILIARY ACTION AND ATELECTASIS

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THE physiology and physics of the movement of oxygen and other gases in the lungs seem to be well understood. The partial tensions of the gases in alveolar air have been measured, and the observed physiologic steps are consistent with the known laws of physics. Removal of entrapped air under pathologic conditions in the lungs and elsewhere is not so completely understood. For instance, it seems to be generally believed that the air from a lung portion which becomes atelectatic is removed by absorption alone. It is held that a plug of mucus, of such great viscosity that the cilia are unable to handle it, corks a bronchus and that the air behind it is completely absorbed.[5,9,10,11] Sometimes the opinion is ventured (seemingly without any direct evidence) that ciliary action is subnormal or has ceased entirely.

In many respects, this view appears to be essentially correct. There seems to be no doubt that air can be removed from an obstructed lung by absorption alone. This has been demonstrated experimentally[1,2,3] and seemingly happens in the presence of tumors and some foreign bodies. There are, however, discrepancies which make one suspect that other factors are involved. Pathologists and endoscopists sometimes find masses of very viscid mucus, but, more often, it is soft. The negative pressure associated with atelectasis may be considerable—34 mm. of mercury has been measured. It is sufficiently strong to move the mediastinum to one side or the other and to practically immobilize the chest wall. One would suppose that the soft mucus would slide down into the area of negative pressure, but this it does not do. The absorption of air experimentally requires sixteen hours,[1,2,3] and postoperative atelectasis develops much more rapidly than that; surgeons have told me that it develops on the table. Thick, viscid casts of mucus, which are incorporated in the bronchial wall, do form in asthma and are demonstrable at necropsy. The result, however, is not atelectasis but rather the opposite, emphysema. That cilia cannot handle viscid mucus appears to be in error also. Tests which I made in the open frontal sinus of an anesthetized dog indicated that viscid mucus can be removed more readily than very thin mucus. In these experiments, it was lifted en masse from the bottom of the sinus and carried to and through the ostium, occluding the latter completely during its passage.

It has been taught that the bronchial tree increases its cross-sectional area with each subsequent branching and that a composite bronchial tree would look like an inverted funnel. If this concept were accurate it might be possible that a viscid plug of mucus, carried upward in the bronchial tree, would become lodged in the narrowed stream bed. My son and I made some measurements, however, of the bronchial tree which indicated that the concept of the inverted funnel applied to the bronchial tree is not correct, at least in bronchi larger than 1.5 mm.

The question arises whether, according to the known laws of physics, it would be possible for the air to be completely absorbed from an obstructed lobe subjected to a negative pressure of the magnitude found in atelectasis.

It may be of value to review the steps by which air is absorbed from different portions of the body in the light of known physical laws. We naturally think of the lungs first. The tidal respiratory flow while at rest is about 500 c.c.; 150 c.c. of each breath remain in the dead space of the bronchial tree; the other 350 c.c. flow into a residual volume of 2,500 c.c. Here a rather complex situation is set up due to the differences in the partial gas tensions in alveolar air and atmospheric air. Diffusion is a necessary step in bringing the fresh oxygen to the alveolar wall where exchange of gases with the blood takes place. Oxygen molecules move at the rate of about 200 meters per second at room temperature; however, they bump into other molecules so frequently that the diffusion rate is very much smaller.

A physicist friend has given me the following formula for the diffusion of air at standard conditions of temperature and pressure: $r = 3.5 \times$ the square root of t ("r" represents the average mean distance in centimeters traveled by the molecules as though they emerge from a point and move outward in all directions without obstruc-

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tion and "t" is the time in seconds). Working this out, r = about 35 mm. per second. The alveoli are approximately 0.2 mm. in diameter or 0.1 mm. in radius. This means that, during the

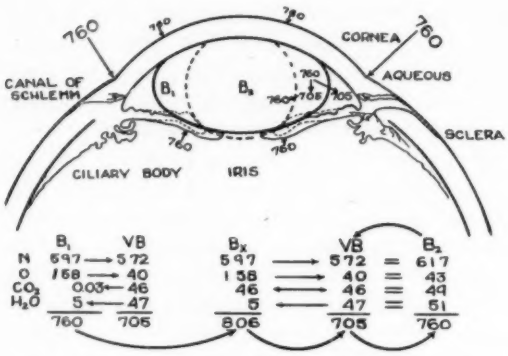

Fig. 1. Diagram representing an air bubble injected into the anterior chamber of the eye. The figures in the column under B_1 are the partial pressures of the various gases contained in the injected air. Column VB gives the values in venous blood. It is assumed, for the purposes of this study, that the values are the same in the aqueous humor of the eye, although they probably differ somewhat. Since CO_2 diffuses more rapidly than the other gases, this gas would pass from the aqueous into the bubble faster than O_2 would dissolve. Therefore, at first, the pressure would increase, as indicated in Bx, if the volume should remain unchanged. The exchange of the other gases would soon take place as indicated between Bx and VB. Equilibrium would be established at 705 mm. of pressure—if the volume should remain constant. The volume, however, does not remain constant since the yielding walls of the eye are subject to an atmospheric pressure of 760 mm. The volume would shrink until the pressure was equalized. Then the gas tensions would all be too high, as in B_2, and movement of molecules would continue into the aqueous, again causing a further fall in pressure. Equilibrium could not be attained and all of the air would be absorbed.

one second which the air remains in the alveolus during inspiration, an oxygen molecule starting from the center of an alveolus could make the trip to the wall about 350 times. The picture then of the gases from the inflowing air (oxygen, carbon dioxide, nitrogen and water vapor) is that of the contained molecules flying rapidly in all directions and making contact with the walls of the alveolus from 1 to 400 times a second. The rate of absorption into the blood would depend upon, first, the nature of the interposed membranes; second, the comparative pressures of the gases in the blood and in the alveolus and, third, the relative solubility of the gases in the blood plasma.

Let us take a simpler example, for the moment, than that of the alveolus. In cataract surgery, we sometimes inject air into the anterior chamber at the end of operation in order to prevent adhesions between the iris and the incision. From four to six days are required for such a bubble in the anterior chamber to be absorbed. Let us follow the steps as we feel they must be, according to physical law (Fig. 1).

The partial gas pressures in the atmosphere are given about as follows: oxygen 158 mm. of mercury, carbon dioxide 0.03 mm., nitrogen 597 mm. and water vapor 5 mm. (water vapor, of course, varies widely) making a total of 760 mm. of pressure. The partial pressures of the aqueous are between those of the arterial and the venous blood; oxygen is given at 40 to 45 mm. and carbon dioxide about 45 mm. The aqueous is produced in the ciliary body through a combination of dialysis, filtration and secretion. Oxygen, nitrogen and carbon dioxide molecules are roughly about the same size as water and would, therefore, probably pass through the capillary walls, the stroma and the endothelium of the ciliary body about as rapidly as water, and, therefore, the concentrations would be much the same as in arterial blood. (Again, a reservation must be made, remembering that filtration may be interfered with by the processes of secretion). The aqueous serves the metabolic needs of the lens, portions of the cornea and iris and probably other structures as well. In this metabolism, it undoubtedly loses oxygen and picks up carbon dioxide, as in the metabolism of tissues everywhere. It leaves the eye by several different routes, including the canal of Schlemm, through which the aqueous is in direct contact with venous blood with no intervening membranous barrier.

Let us assume, for our purposes, that the gas tensions present in the aqueous are the same as those in the venous blood. Those given for the venous blood are: oxygen 40 mm. of mercury, carbon dioxide 46 mm., nitrogen 572 mm. and water 47 mm., making a total of 705 mm., or 55 mm. less than that of the atmosphere. The following steps would occur during absorption of the air bubble in the anterior chamber: The 0.03 mm. of carbon dioxide would be rapidly increased to 46 mm., because the movement of carbon dioxide is some thirty-five times as fast as that of oxygen. Meanwhile, the 158 mm. of oxygen would be reduced more slowly to 40 mm. The bubble would first increase in size or pressure because of the rapid movement of the carbon dioxide; the nitrogen moves much more slowly than either oxygen or carbon dioxide and, for purposes of simplification, can be assumed to stand practically still. The carbon dioxide, having reached equilibrium before the oxygen, would find itself at a higher tension in the bubble as the latter moved out. If the bubble remained constant in size, the carbon

dioxide would move back into the aqueous and the oxygen would continue to move into the aqueous until equilibrium would be established with a net loss in pressure of 72 mm. However, since the eye is subjected to atmospheric pressure, disregarding for our purposes the intraocular pressure, the air bubble would not remain the same in size but would shrink until the pressure again equalled 760 mm. As soon as this happened, then the tension of the carbon dioxide, oxygen and the nitrogen would be greater in the bubble than in the aqueous, so all three would again move from the bubble into the aqueous—the carbon dioxide very rapidly, oxygen much more slowly and the nitrogen still more slowly. This would once more be followed by shrinkage of volume and the whole process would be repeated. Equilibrium would never be established, and eventually all of the gases in the bubble would be dissolved in the aqueous.

Taking another example from our field of work, negative pressure sometimes develops within the sinuses. Assuming that the ostium should become completely blocked while the sinus was still full of air, through what physical steps would the gases of the air pass and what would be the eventual pressures? Conditions in the sinuses are far different from those in the alveolus of the lung or those in the anterior chamber of the eye. In the eye, the air is in direct contact with the aqueous, there being no interposing membranes. In the alveolar wall, there is at least one interposing membrane—the capillary wall. In the sinus, there is the capillary wall, a connective tissue stroma and a cuboidal type of epithelium, which is at least two cells deep. It would seem that these interposing structures would increase the time of the passage of gas molecules from the interior of the cavity into the blood stream. Moreover, the blood supply to the sinuses is normally very meager, and still another factor is that the sinus wall carries both oxygenated arterial blood and venous blood, while all of the blood entering the lungs is venous blood, from the standpoint of gas content.

Despite these handicaps, carbon dioxide would eventually find its way from the capillaries into the sinus cavity, oxygen would move less rapidly from the cavity into the venous blood, and, still more slowly, nitrogen would find its way into the blood stream. The total pressure within the sinus would rise at first because of the inflow of carbon dioxide molecules, but, eventually it would begin to fall, as was the case in the eye. However, the sinus walls being rigid, there would be no decrease in volume and, therefore, the pressure, after the initial rise, would fall progressively until equilibrium would be established. If there were only venous blood present in the capillaries, the pressure theoretically would fall to 705 mm. of mercury, the same as the gas pressure of the venous blood. However, the gas pressure of the arterial blood is 757 mm., therefore, the gas molecules would escape from the arterial blood into the cavity as they were removed from the cavity into the venous blood. Theoretically, equilibrium should be established at a pressure somewhere between 705 and 757 mm., probably in the neighborhood of 725 mm. The volume would remain the same as when closure of the ostium began (excluding, of course, such things as edema and secretion).

As a further illustration, the middle ear furnishes an example of conditions between those in the eye and in the sinus. Herbert,[6] of Upsala, on introducing rubber balloons into the nasopharynx and inflating them in such a way as to close the eustachian tubes, has found that in about thirty minutes there is a marked retraction of the ear drum, which he assigns to the reduced pressure following oxygen absorption. Here are conditions midway between those in the eye, where there is an external positive pressure and yielding soft walls, and those of the sinus, where the walls are rigid and there can be no reduction in volume. That portion of the lateral wall of the middle ear which is comprised of the tympanic membrane is more or less yielding and the volume of the space is reduced somewhat. Therefore, the air in the middle ear would be more completely absorbed than that in the sinus. Still it is not possible for all of the air* to be absorbed; equilibrium would eventually be established at a pressure somewhere between the gas pressures of the arterial and the venous blood. One would infer the time required, as in the case of the sinus, would be long compared with that of the lung.

In the thorax, too, the tissues about an obstructed lobe of the lung are not free to yield as completely as those about the bubble in the eye (Fig. 2). The wall of the thorax is more or less rigid and a negative pressure develops within it.

*Again, excluding such things as secretion, edema and other changes in the lining epithelium.

However, absorption can go on until the negative pressure equals the difference between the gas pressures in the alveolus and the gas pressures in the venous blood. This difference approximates

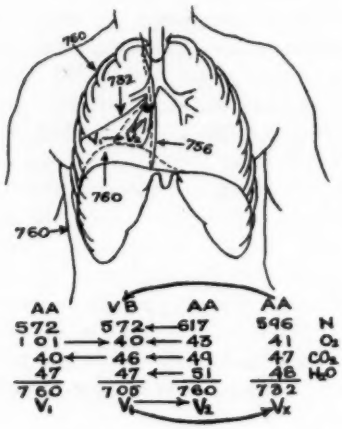

Fig. 2. Absorption of air from an obstructed lobe of the lung and the effect of negative pressure. AA represents the gas tensions in alveolar air and VB the gas tensions in venous blood. When a lobe becomes obstructed exchange of O_2 and CO_2 would take place as indicated between AA and VB. If the volume (V_1) remained unchanged, equilibrium would be established at 705 mm. It would not remain constant, however. It would yield to the pressure of 760 mm. (atmospheric pressure) in the surrounding tissues and shrink to V_2. All of the values would then be too high and gas molecules would pass from AA_1 to VB. If a negative pressure of 732 mm. should develop within the thorax, as a result of shrinkage of the lobe (such pressures have been measured), then there would be further shrinkage in volume until the pressure within the lobe was also 732 (Vx). The gas tension would still be somewhat above venous blood and would move from AA_2 to VB. Equilibrium would not be possible unless the negative pressure within the thorax should drop to 705 mm. (venous blood) or below. If the intrathoracic pressure should remain above 705 mm., theoretically absorption could be complete.

55 mm. of mercury. Theoretically then, there could be established a negative pressure of 55 mm. of mercury before equilibrium would be established and the absorption of gases would cease. The greatest negative pressure which has been measured in collapse of the lung is about 34 mm. of mercury,[4] so, theoretically, all of the air from an obstructed lobe of the lung could be completely absorbed, provided 55 mm. of negative pressure is not exceeded. However, a certain amount of time is required for absorption of gases, even from the lung. Coryloss and Birnbaum[1,2,3] report experiments in which individual gases were injected into the obstructed lung of a dog (i.e., a portion of the lung had been tied off). Absorption was accomplished for carbon dioxide in four minutes, oxygen fifteen minutes, nitrogen sixteen hours and air sixteen hours. As these experiments indicate, nitrogen and air require about the same amount of time for absorption.

Attempts were made to compare the absorption rate from the lung with that from the anterior chamber of the eye. The volume of the anterior chamber of the eye has been estimated to be about 1/20,000 of that of the lungs. The volume of blood flowing through the lungs at rest is estimated at about 4,600 c.c. per minute; through 1/20,000 part of the lung, this would be 230 cubic millimeters. The rate of circulation of aqueous through the eye is unknown; measurements have been variable (from 2 to 40 cu. mm./minute) but even the maximum would be only a small fraction of the 230 cu. mm. in the lungs. There are several other factors which make comparison difficult. Although there is no interposing membrane between the bubble and the aqueous in the eye, the bubble is all in one mass and exposes the minimum of surface for absorption, whereas an equal volume of air in the lungs is divided into hundreds of little spheres, each of which is surrounded by blood flow, presenting an area of about 500 square centimeters. In the aqueous, there are no blood cells to aid in the quick pick-up of the gas molecules, as there are in the blood surrounding the alveoli. It is difficult also to compare the time of absorption in the sinuses and ear with that in the lungs; in the former the air masses exhibit a small area for absorption compared with the latter. Moreover, the blood flow is only a small fraction of that in the lungs and the interposing membranes are very much denser and thicker. If sixteen hours are required to absorb a volume of air from an obstructed lobe of a lung, probably several days would be required to absorb an equal volume from an obstructed sinus.

To summarize, air injected into the anterior chamber of the eye absorbs completely, but the time is comparatively long; it takes four to five days to absorb 0.1 c.c. It requires several days for air to be completely absorbed from the pleural cavity or the subcutaneous tissue and several weeks from the peritoneal cavity. The only experiments bearing directly on the time factor in the case of the lungs, which I have seen reported, are those by Coryloss and Birnbaum, where sixteen hours were required to absorb air from the normal lung of a dog. These facts concerning the time of absorption would seem to indicate that

some factor in addition to absorption is acting in the removal of air in postoperative atelectasis, which develops very promptly after operation.

In all of the articles in the literature on postoperative atelectasis, the matter of ciliary action is almost entirely overlooked. It seems to me that ciliary action cannot be ignored in the respiratory tract any more than heart action can be disregarded in the circulation or peristalsis in studying the physiology of the gastrointestinal tract. There is no evidence that ciliary action in postoperative atelectasis is subnormal.

Some years ago I performed some experiments on the tracheas of freshly killed hens for the purpose of determining a possible relationship between ciliary action and postoperative atelectasis. If an occluding mass of mucus is introduced into the lower end of such a trachea, it will pass through to the upper end by ciliary action in the course of a few minutes. In these experiments the lower end was connected to a water manometer immediately after introducing the mucus; as the mucus advanced toward the laryngeal end, by ciliary action, a negative pressure developed rapidly, reaching a maximum in less than twenty minutes. This experiment was done on nineteen tracheas with the same result in all. The recorded negative pressures varied from 5 to 40 mm. of water.

In order to be certain that this phenomenon was not due to absorption of oxygen in the fresh tissue, another series of experiments was performed connecting the laryngeal or upper end of the trachea to the manometer. In each of these instances a positive pressure developed of about the same magnitude, namely up to 40 mm. of water. Having in mind the probability that many masses of mucus in the longer tubes of a patient suffering from atelectasis might produce a cumulative effect, resulting in a higher pressure, another series of experiments was performed.[8] Three, and sometimes four, tracheas were arranged in tandem and each was connected to a water manometer at its upper end. Connections which could be opened and closed at will joined the tracheas so that the pressure resulting in each could be individually measured, and, when desired, all of them could be connected as a single tube and the cumulative pressures recorded. These experiments demonstrated clearly that the effect is cumulative and pressures as high as 150 mm. were obtained in this way. This is comparable to the pressures measured in postoperative atelectasis.

Several series of experiments were done on the frontal sinuses of dogs. In the first of these, two needles were forced into the frontal sinus of an anesthetized dog, one was connected to a water manometer and through the other a quantity of mucus was injected. A negative pressure began to develop after a few minutes, reaching a maximum in about twenty minutes; some of these pressures were as high as 60 mm. of water. In order to rule out the factor of absorption, a second series was done in which the dog was bled to death through the femoral artery in the middle of the experiment, and then decapitated. Neither procedure caused any significant variation in the pressure. Still other experiments were done on the decapitated heads of freshly killed dogs. These recorded the same phenomenon of a rapidly forming negative pressure.

These experiments on the trachea of the hen and the sinus of the dog demonstrate another mechanism for the removal of air and the development of negative pressure. It is essentially a piston-cylinder action motivated by ciliary power, the masses of mucus acting as the pistons. If this factor is acting in the development of postoperative atelectasis, we would have the explanation for the failure of the soft mucus to slide into the area of negative pressure and for the seeming inaction of the cilia. When the cilia have pushed the masses of mucus up a bronchus as far as they are able, against atmospheric pressure, the mass of mucus becomes stalled. This explains the fact that very soft mucus can seemingly act like a cork; the cilia are holding it in position and attempting to push it further. The negative pressures of 200 to 400 mm. of water, which have actually been measured in postoperative atelectasis, might readily be explained on the basis of a series of mucus pistons in tandem being pushed upward in the bronchial tree by ciliary action. This negative pressure could, theoretically, be produced entirely by ciliary action independent of absorption. The negative pressure is probably maintained by ciliary power only.† One can say that the effec-

†There is another mechanism possible which should be mentioned. If the occluding mucous piston occurs in a membranous bronchus, it is quite possible that the bronchus may collapse behind the piston, due to the negative pressure. If this were to happen, the apposing walls of the bronchus might adhere together because of the cohesiveness of the normal mucous film. In this case, the atelectasis might conceivably be maintained without the presence of an abnormally great quantity of mucus.

tive power of the cilia equals the atmospheric pressure minus the pressure in the affected lobe and is the magnitude of the negative pressure. This pumping action of the ciliary mechanism is similar in principle to that of a mercury vacuum pump.

It was found in the experiments on the trachea of the hen that if the procedure were carried on for an hour or more the cilia continued to whittle away at the periphery of the mucus piston, gradually carrying it upward and depositing it at the end of the trachea. Theoretically, the cilia could remove the piston maintaining the negative pressure in atelectasis also, if given sufficient time, provided no more mucus formed. However, if production of excessive mucus should continue, then the atelectasis might be maintained.

We have spoken of absorption of air from the sinuses as though it were the usual thing for the ostium to be obstructed while the cavity is still full of air. It is more likely that in conditions resulting in vacuum headache the air was initially displaced more or less completely by the first secretion which formed. It seems likely that the heavy mucus, which forms large viscid masses toward the end of an attack of sinusitis, might produce a negative pressure in a manner similar to that in the experimental dogs. This could very readily be the explanation for the clinical condition known as vacuum headache.

This last suggests another possibility for the rapid removal of air from the lobe of a lung in a surgical patient during operation. It is quite possible that the air is largely displaced by a secretion and that a negative pressure soon follows due to ciliary action.

The negative pressure which occurs in the middle ear might very likely be on the basis of ciliary action moving pistons of mucus down the eustachian tube. Repeated attempts to demonstrate it experimentally have failed to date. Just how the air is removed from the middle ear in blockage of the eustachian tube remains an open question.

Summary

The absorption of air from the anterior chamber of the eye, from the sinuses, from the ear and from obstructed portions of the lung follows the principles of well-known laws of physics such as those governing diffusion, solubility, partial pressures and molecular combination. It seems that air can be completely absorbed from almost any space or tissue in the body, such as those just mentioned, as well as the peritoneum, pleural cavity and subcutaneous tissues, provided sufficient time is allowed and provided the space containing the air can collapse. If the space containing the air cannot collapse or can do so only partially, a negative pressure develops, depending upon the gas pressures present in the absorbing fluid.

In postoperative atelectasis, there are at least three mechanisms by which the air could be removed, namely, (1) absorption, (2) displacement by secretion, and (3) the pumping action of cilia and moving masses of mucus. The negative pressure in most cases of postoperative atelectasis is, in all probability, maintained solely by ciliary action. It is probable that the mechanism involved in the pumping action of the cilia and moving masses of mucus is the one which causes vacuum headache and possibly also negative pressure in the middle ear following otitis media.

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TRANSFUSION PROBLEMS

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DURING the past twenty years there has been a remarkable increase in the use of blood transfusions. In one hospital with which I am connected the increase has been exactly twenty-fold. There are many reasons for this striking increase. The changes in surgical methods and the improvements in anesthesia together with the development of the antibiotics have made surgical procedures commonplace which a decade ago were rarely done. Generally this type of surgery necessitates the use of a large amount of blood. The introduction of the ACD solution as a preservative permits the storage of blood for periods of about three weeks, and contributes greatly to making blood more available and hence used more often. Probably the greatest factor in promoting the increased use of blood is the safety of the modern transfusion. The discovery of the Rh factor initiated a tremendous amount of research which has for the most part eliminated the frequent and severe transfusion reactions which were so common prior to 1940.

The safety of a transfusion and its frequent use paradoxically creates a problem. The use of blood is so ordinary and so commonplace that there is today a tendency to look upon a transfusion as a benign procedure much like the administration of saline or glucose. A transfusion always carries with it a definite danger. Disrespect for the lethal possibilities involved can and still does lead to tragic results. Let me cite one example.

In one hospital a unit of blood was ready for an eight o'clock operation. However, it was not used, and it was left in the room after the operation was over. The second operation in this room required a transfusion, and when the anesthetist decided to start the blood she picked up the bottle standing on the side table, and over 400 c.c. was administered before she read the label and realized that that blood was intended for the prior surgical patient. We must continuously teach our hospital personnel the tragic results which may come from carelessness and negligence in the use of blood.

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Another problem evolves around the question, "Does the patient actually require a transfusion?" Physicians sometimes forget the normal range of hemoglobin. In this section of the country the lower limit of average hemoglobin is about 12 grams. This means that 70 per cent on a 17 gram standard is a low normal. It is very probable that hemoglobin is adjusted by the body to the particular activity and physiology of the individual. A hemoglobin of 13 grams in one individual may be just as normal as 16 grams in another. If blood is transfused into such an individual having a physiologically normal hemoglobin of 13 grams, that blood is an excess and cannot be utilized and will be eliminated by hemolysis.

There is a tendency today to use a transfusion as an accessory to many operative procedures. Individuals undergoing operations of modern magnitude, for example a cholecystectomy, are not necessarily candidates for a transfusion. Prior to the operations, these same individuals, being in good nutrition and having no anemia, could have been used as blood donors and as such would have lost 500 c.c. of blood. Surely in such individuals an operation with a blood loss of 200 to 400 c.c. does not necessitate a transfusion except perhaps for psychological reasons. It would seem that a transfusion is too dangerous to be used as a dramatic accent in the care of a patient or a patient's relatives.

On the other hand, there are occasions where there has been significant or massive loss of blood, and transfusion is the life-saving therapy. When such occasions arise, the treatment with blood must be prompt and must be generous. Administration of one unit of blood over a period of twenty to forty minutes to an invidual bleeding critically is not adequate treatment. Two, three, or four units of blood should be given simultaneously using each arm and leg as portals, or preferably, the blood should be forced in by pressure. When a patient is losing blood rapidly it is probably impossible to replace the blood too fast. Five hundred c.c. of blood can be given by pressure in four to seven minutes. There should be no hesitation in giving two, three or more units in suc-

cession as long as there is evidence that the patient is bleeding.

Every hospital should have available in surgery, in obstetrics, and in its supply room the equipment for giving blood under pressure, and everyone should know how to use it. Most of the commercial companies handling transfusion equipment now have such pressure sets for sale, and their people are happy to give instructions as to its use and the precautions to take.

In hospitals without a large blood bank, the selection of the kind of blood to use during an acute emergency presents a difficult problem. For example, should time be taken to do Rh typing? Today, because of the reliable typing sera now available Rh grouping can be done in a few seconds. It would be a rare situation wherein blood must be given without knowledge of the Rh grouping of the recipient. If the patient is Rh positive, the problem is simple because blood of either Rh type can be given. The question of sensitization to the Hr antigen in Rh-negative blood can be disregarded because of its great rarity. If the patient is Rh-negative and a sufficient amount of Rh-negative blood is not immediately available, it is necessary to pause for a few moments and evaluate the situation. If the patient is a male, the question must be asked, "Has he been transfused before?" If so, he may have been sensitized by this prior transfusion, in which case these questions must be asked: "Is the emergency great enough to warrant taking any chance on a prior sensitization? Can the emergency be controlled by plasma until Rh-negative blood is available?" The physician must balance the urgency against this risk. If the patient is an Rh-negative female, the problem is much more difficult. Because of the very adverse effect on future pregnancies, no female should ever be deliberately sensitized by a transfusion of Rh-positive blood except as a life-saving measure. Any Rh-negative woman who has had children may be sensitized, and if her obstetrical history suggests that she may have had erythroblastotic babies she most likely is sensitized. Transfusion of Rh-positive blood to these sensitized Rh-negative women results in extremely severe reactions with a very high mortality. In all such cases of possible sensitization, plasma must be used until Rh-negative blood can be obtained. Every community should make arrangements so that the Rh-negative individuals in its population are known and have been organized so that they may act as donors for each other in emergencies.

The use of Group O blood as a universal blood for transfusion does carry with it a certain small chance of reaction. An instance of this type was studied recently by Dr. Matson and will be reported in MINNESOTA MEDICINE. However, the incidence of this type of reaction is very small, and a patient's life should never be endangered by hesitating to use Group O blood for other groups and to do so, if necessary, without cross matching. The addition of the Witebsky substance to such Group O blood takes only a few seconds and still further lessens the danger. The material is purified Group A and Group B blood substance. When this is added to Group O blood, the anti-A and anti-B agglutinins are neutralized, making the blood a truly universal blood.

As a general pathologist, I have had contact with transfusion work for many years and I know that we never had such transfusion safety as we now have. Most of this safety is due to the study and research of men who are devoting their full time to transfusion problems. I am sure that the practitioner owes such men considerable gratitude and support.

THE MANAGEMENT OF STATUS ASTHMATICUS

(Continued from Page 987)

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History of Medicine In Minnesota

MEDICINE AND ITS PRACTITIONERS IN OLMSTED COUNTY PRIOR TO 1900

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(Continued from September issue)

Seth (Septimus) Watkins Gould (1847-1891), a native of Canada, was a resident of Olmsted County the greater part of the time from about 1873 into 1891. There is some evidence that he was in Oronoco, this county, in 1873, and that during 1874 he was in Zumbrota, Goodhue County. He probably came to Rochester in 1875, for on April 21, 1876, the Rochester *Record and Union* stated that Dr. Gould, who for some time had been associated with Dr. W. W. Mayo, had gone to reside and to practice medicine in Mazeppa, Goodhue County. In July, 1880, well liked, considered to be a capable practitioner, he returned from Mazeppa to Rochester, again to be an associate of Dr. Mayo. The agreement between Dr. Mayo and Dr. Gould was dated July 24, 1880, and was witnessed by William J. Mayo and W. Logan Brackenridge. It was stated in the newspapers at that time that Dr. Gould was a graduate of Rush Medical College, class of 1866, and that in addition to the duties of an associate physician he would have charge of all patients requiring the services of an electrician, that he had made a successful study of electrical therapeutics. The first official register of physicians of Minnesota (1883-1890), however, listed Dr. Gould as a graduate of the Bennett Eclectic Medical College of Chicago in 1870; he was licensed in Minnesota on December 31, 1883, when he received state certificate No. 665 (E).

During his second period of practice in Rochester, it is said, Dr. Gould was married to Miss Oaks of that place. From May, 1881, until late December, 1881, when he removed to Pleasant Grove, he was secretary of the Rochester Board of Health, during the presidency of Dr. E. W. Cross. In Pleasant Grove Dr. Gould bought the property of a Mrs. Hill, established a drug store and entered his final practice of medicine. His professional contemporary in the village was Dr. Alonzo W. Hill, who practiced there from 1878 to 1889.

On January 1, 1886, Dr. Gould, thirty-nine years of age, was committed by his wife to the Second Minnesota Hospital for Insane at Rochester. The origin of his illness was ascribed locally to sunstroke suffered while working over a gun during his military service (reputedly as a lieutenant during the Civil War), and it was said that a second soldier, working with him, also succumbed to sunstroke and became insane. Dr. Gould died in the state hospital on October 27, 1891, from "general paresis with asphyxia," and was buried in Oakwood Cemetery, Rochester.

Christopher Graham (1856-), venerable physician emeritus of Rochester, a practitioner of medicine from 1894 to his retirement in 1919, arrived in Olmsted County early in July, 1856, when he was three months old.

The sixth of the thirteen children of Joseph Graham and Jane Twentyman Graham, he was born on April 3, 1856, near Truxton, Cortland County, New York. His parents were natives of Cumberland County, England; Joseph Graham

was born near the village of Dalston, Jane Twentyman in Great Orton Parish. The Twentyman family came to America in 1840; Mr. Graham came in 1844 when he was twenty-two years old. Mr. and Mrs. Graham were married in Cortland County in 1847 and lived there until May, 1856, when with their six small children and a few worldly goods in a horse-drawn wagon they started for Minnesota. Although heavy rains forced them to take a boat from Buffalo to Milwaukee, they finished their journey by wagon into Kalmar Township, Olmsted County, and there founded a new home about five miles northwest of Rochester. After forty years at Grahamholm, which they had first called "The Willows," they removed to Rochester to spend their last years. Their thirteen children were: Mary Elizabeth, William Beck, Thomas Chambers, John, Manfred Davis, Christopher, Joseph, Dinah Frances, Margaret Anne, Frank Charles, Edith Maria, Arthur Frederick and Jennie. In 1949 there were living, Joseph Graham, on a farm near Stewartville, and Dr. Christopher Graham, of Rochester.

The story of the Graham family should be chronicled as a saga and the group commemorated for intelligence, fortitude, integrity and good citizenship. Under the severest hardships of pioneer days parents and children surmounted lack of means and opportunity. Joseph Graham was a kind father, although a strict disciplinarian in the home, and an indefatigable worker who had unusual capacity for taking pains. Jane Twentyman Graham was a woman of rare courage, deep religious faith, warm sympathy and a natural talent for soothing the sick and making them comfortable. Always giving, never asking, as since has been said of her physician son, she nursed without material recompense all in her vicinity who needed her. Perhaps she gave greatest aid in obstetrical cases, in which her knowledge and skill were equal to those of most physicians of the day. In later years her children estimated that she aided in the birth of 243 babies, without medical counsel and without loss of a mother or a child. Two of her daughters, Dinah Frances and Edith Maria (Mrs. Charles H. Mayo) inherited her natural ability in nursing; Dinah Frances, the elder, in her early teens helped her mother in cases of all types. Both of these daughters received accredited training and followed the profession of nursing before marriage. Edith Graham was the first trained nurse in Rochester.

It will require an abler pen and a more suitable vehicle than the present to portray rightly the life of Christopher Graham. Under conditions so difficult that today they are hard to picture, he obtained his education by patient, persistent effort. Until he was twenty years old he worked on the home farm, as did his brothers and sisters, and received only the teaching available in the local district school during terms of a few weeks at most. Books were few, money was too scarce to buy them, and sense of responsibility forbade borrowing them. In the winter of his twenty-first year "Kit" Graham achieved four continuous months of study at the private school of Mr. Loofborough, in Rochester; he then had covered approximately the work of the present day junior high school. The next winter he taught district school and in the following year studied a full term of nine months at Niles Academy (The Rochester English and Classical School, staffed by Mr. and Mrs. Sanford Niles and their assistants). The next four years were filled with farm work, rural school teaching and, in spring and autumn, further study at Niles Academy. Always Christopher walked from home to school and back.

In the autumn of 1882, aged twenty-six years, Christopher Graham entered the University of Minnesota as a subfreshman and special student. On arrival, doubtful of his eligibility, he told President William Watts Folwell of his meager schooling and explained that he probably could attend the university only a year or so, that he loved domestic animals and the land and wanted to be a farmer.

President Folwell assured him that his lack of formal preparation was not a drawback, that his age was an advantage. For the ensuing five years he was a student at the university, dropping out frequently to repair his finances. Thereafter, for two school years, from 1887 into 1889, he taught chemistry and natural philosophy at Shattuck School at Faribault. In the autumn of 1889 he entered the class of veterinary medicine at the University of Pennsylvania and in June, 1892, took his degree; it was then that he received the Lippincott Prize, awarded to the member of the graduating class who had attained the highest general average in the course of three years. After one year as veterinarian to the Experiment Station and Agricultural College of the University of Minnesota, he realized that clinical medicine was his true calling, and he returned to the University of Pennsylvania as a medical student. When in June, 1894, aged thirty-eight years, he received his degree of doctor of medicine, he returnd at once to Rochester, to accept the invitation of Drs. William J. Mayo and Charles H. Mayo to join them in practice. These brothers he has credited with giving him encouragement and opportunity.

The following twenty-five years saw his fulfillment as a physician. He was the first to serve as intern in St. Mary's Hospital (opened in 1889) and he became and remained an attending physician. Throughout the years he studied and observed, visiting hospitals and clinics at home and abroad and taking special courses of study, particularly with regard to the blood. In his earlier period he was an obstetrician of ability; today mature practitioners of this specialty who studied as young physicians with Dr. Graham, express their debt to his wisdom and conservatism. Gradually he fixed his chief interest on diseases of the digestive tract and their differential diagnosis and here he achieved his most brilliant success. The papers which he contributed to the medical literature on chronic appendicitis, gastric ulcer and gastric cancer and gallbladder disease retain their value. His paper on disease of the gallbladder, written before the perfection of certain laboratory tests and the initiation of roentgen examination of the gallbladder, is a classic in diagnosis by case history, signs and symptoms. Dr. W. J. Mayo once said of Dr. Graham: "I have never met his equal as an internist and a diagnostician." Senior associates and junior, alike, recall Dr. Graham as at all times modest and unassuming, distinguished for his insight and acumen, sympathy, patience and delightful humor.

Dr. Graham early became a member of the Olmsted County Medical Society (once its president), the Southern Minnesota Medical Association, the Minnesota State Medical Society and the American Medical Association, and was active in them throughout his professional career. Interested in public health and sanitation, he was a member of the state board of health for several years during the administrations of Governor John A. Johnson (1904-1909), and retired from the work only because of professional duties in Rochester. He was appointed associate in medicine with the Drs. Mayo in 1904 and head of the Division of Medicine of the Mayo Clinic in 1914. He was Professor of Medicine on the Mayo Foundation for Medical Education and Research of the University of Minnesota from 1915 until his retirement in 1919. He is a member of the Alumni Association of the Mayo Foundation.

Christopher Graham's love of farming, horticulture and animal husbandry increased with the years, leading him to distinguished achievement and honor in these fields. He was a leader in introducing Orpington chickens into the country, and at one time had flocks of these fowls, black, white, and buff, second to none in the world. He started the development of what was probably the first purebred Holstein-Friesian herd in this part of the state and he became internationally known

as a breeder and importer of blooded livestock. For more years than any other person he was a member of the board of trustees of the Holstein-Friesian Association. His scientific contributions have been recorded in the archives of the Minnesota Livestock Breeders Association and elsewhere.

Dr. Graham is a Mason and an enthusiastic member of the Rotary Club. The latter organization, he has said, has helped him more than has any other influence to understand men and to obtain a broad comprehension of social and civic needs and responsibilities. His support of the Olmsted County Fair Association and his generous gifts of real estate to that association and to the city of Rochester and St. Mary's Hospital evidence his constructive interest in public welfare. He is a supporting member of the Calvary Episcopal Church.

On January 4, 1899, Christopher Graham was married to Elizabeth Blanche Brackenridge, member of a family long notable in Olmsted County and the state. Walter Lowry Brackenridge, of Scotch ancestry, a lawyer, came in 1856 from Pennsylvania to Rochester, Minnesota, with his wife, Margaret McC. Logan Brackenridge. The three children of the family were William Logan, Madge and Elizabeth Blanche; the son, who died in 1905, was a lawyer in Rochester; Madge was married to George D. Parmalee.

For many years Dr. and Mrs. Graham have made their home in East Rochester, in the beautiful brick residence, surrounded by parklike grounds, that was built by the Honorable Walter L. Brackenridge in the early seventies. They have two children, Malcolm Brackenridge Graham and Elizabeth Blanche (Mrs. George M.) Lowry, both of Rochester, two grandchildren, Margaret Brackenridge (Mrs. Calvin T.) Slatterly and Louise Lowry, and two great-grandchildren.

The esteem in which Dr. Graham is held by his fellow citizens cannot be measured, but it is indicated by an inscribed scroll that was presented to him on his eighty-fifth birthday on April 3, 1941:

> Diplomate in both veterinary and regular medicine, authority on diseases of the upper part of the gastro-intestinal tract, practical farmer, patron of animal husbandry, public-minded citizen, unannounced doer of good to scores of his fellowmen, on this the occasion of his eighty-fifth birthday, his many friends and townsmen present this scroll in token of affection and esteem.

Charles Topliff Granger (1870-1939) practiced medicine in Rochester, Olmsted County, from 1892 into 1939, with the exception of two years in McGregor, Minnesota, from 1928 to 1930. For thirty-five years he had his office over the Quale Drug store on Broadway.

Born on July 30, 1870, at the farm home of his parents in Cascade Township, Olmsted County, near Rochester, Charles T. Granger was the second son of Abner Granger and Louise Topliff Granger. Abner Granger was the son of Julius Granger and grandson of Seba Granger, who was descended from a Granger who settled in New England long before the American Revolution; Seba Granger removed from Massachusetts to Otsego County, New York, and there established the family home. Abner Granger was married to Louise Topliff, of Otsego County, on February 25, 1867, and in that year came with his wife to Olmsted County, Minnesota, where for many years he was a highly respected citizen and a substantial farmer and pioneer dairyman.

Charles T. Granger received his early education in rural schools and in the schools of Rochester. He spent a year in the medical department of the University of Iowa and two years at the Hahnemann Medical College, of Chicago, from which he was graduated on March 23, 1892. Returning immediately to Rochester, he entered into partnership with Dr. Wilson A. Allen, in offices in the Leland Block,

on Broadway. At first he devoted himself to treatment of diseases of the eye and ear, but proceeded soon to the general practice for which he became well known. In the autumn of 1892 Dr. Allen and Dr. Granger established the Riverside Hospital, in East Rochester; this institution functioned three years.

From the summer of 1896 Dr. Granger practiced alone for eight years, extending his practice from the city into the county and adjoining counties, until it became, by 1904, according to the *Rochester Post and Record* of June 24, that year, "probably the largest individual practice of any physician outside the Mayos." In the earlier years, before automobiles, it sometimes required four consecutive days of driving to make his rounds.

In 1904 Dr. Granger took into partnership one of his former students, Dr. George T. Joyce, newly graduated in medicine from the University of Illinois, and like himself a native of Olmsted County. After this association ended, Dr. Granger for many years maintained a corps of assistant physicians, replacing them as, after a year or two of work with him, they went on to independent practice.

In his earliest years as a physician Dr. Granger was active in the Minnesota State Homeopathic Institute. After 1900 he was a member of the Olmsted County Medical Society (its secretary, 1905; its president, 1906-1907); the Southern Minnesota Medical Association, the Minnesota State Medical Association, and the American Medical Association. In 1935 he published in the *St. Paul Dispatch* a series of articles, *The Saga of a Country Doctor,* which attracted much attention. Although he refused the nomination for county coroner on the Republican ticket in 1896, he served from 1894 to 1899 as county physician for the city of Rochester and the townships of Rochester, Marion, Haverhill and Cascade. He was active in the Methodist Church (he later became a convert to the Catholic faith, it is said), and in civic affairs, serving as alderman, alderman-at-large, mayor, president of the city council two terms, and as member of the city library board.

On March 8, 1898, Charles T. Granger was married to Katherine Cornelie, of Minneapolis. Mrs. Granger, a woman of outstanding personality and ability, was a trained nurse who for a year or more had been the supervisor of nursing at the Riverside Hospital. Dr. and Mrs. Granger first lived in East Rochester, later in the southwestern part of the city, in a home known for its gracious hospitality. In 1928 they removed to McGregor, Minnesota, where for two years they conducted their own clinic and hospital. Mrs. Granger died in McGregor on March 7, 1929, survived by her husband and by four children, Louise, Virginia, Charles and Gordon. Dr. Granger was married on May 2, 1930, to Bertha P. Irish, widow of Dr. H. R. Irish, of Forest City, Iowa; Bertha Irish Granger, a former resident of Rochester, was a graduate of the nurses training school, since discontinued, of the Rochester State Hospital.

In 1930 Dr. Granger resumed the practice of medicine in Rochester, limited by his gradually failing health. He died in Rochester on October 4, 1939, at the home of his niece, Ophelia Granger (Mrs. E. D.) Ridgeway, survived by his wife and his four children and by his sister, Kate E. Granger, of Rochester. His brother, the Honorable George W. Granger, a distinguished attorney of this city, had died a few months previously. In an obituary in the *Minneapolis Tribune* of October 6, 1939, Dr. Granger was credited with having diagnosed the first case each of epidemic infantile paralysis, Spanish influenza and pellagra in Minnesota, and the first case of trichinosis in Rochester.

In 1945 there were living of Dr. Granger's immediate family: Mrs. Granger, in Rochester; Louise Granger (Mrs. Edward B.) Lynch, in Minneapolis; Virginia Granger (Mrs. Raymond T.) Busch, in Gaylord; Charles T. Granger, a news-

paperman, of Milwaukee, Wisconsin; and Major Gordon A. Granger, United States Army Medical Corps, in England.

Dr. Charles T. Granger is remembered for his keenness, cleverness and social charm, and for certain of his hobbies; namely, his blooded saddle horses and racers of the earlier years, among them Gipsy Wilkes and Kentucky Prince, and his hunting lodge, Granger's Camp, in the beautiful Genoa Woods of Olmsted County, where he was host to his friends among businessmen and physicians of Rochester.

Gertrude Booker Granger (1871-1928) was born Gertrude Booker on March 13, 1871, at Quincy, Olmsted County, Minnesota, the daughter of James A. Booker, a native of Brunswick, Maine, and Jane Short Booker, a native of Waddington, New York. She had four brothers, O. W., Frank Daniel, F. A., and W. Allison Booker. After the death of Mr. and Mrs. Booker, in 1887 and 1889, respectively, Frank Daniel Booker went to Brunswick, Maine, to live with an uncle. He received his education in the East and in 1901, a qualified dentist, graduate of the dental department of the University of Pennsylvania, he settled in Rochester, Minnesota, where for many years he was a leading dentist. The other brothers became farmers of Olmsted County.

Gertrude Booker received her early education in the country schools of Olmsted County and at the Winona High School. In October, 1892, she entered the newly established Asbury Methodist Hospital, in Minneapolis, as a student nurse, and in June, 1894, was graduated. The following September she matriculated in the medical department of the University of Minnesota, from which she received the degree of doctor of medicine in June, 1897. Her license, No. 776 (R), to practice in the state, she received from the Medical Examining Board of the State of Minnesota on June 10, 1897. For the next six months, living in Dover, she practiced medicine in the communities of Dover and Eyota. On January 1, 1898, she joined the staff of the Drs. Mayo, Graham and Stinchfield in Rochester, primarily as assistant to Dr. Charles H. Mayo in the treatment of diseases of the eye, ear, nose and throat; a little later she was given charge of the work on refractions of the eye.

On February 14, 1900, Gertrude Booker was married to George W. Granger, of Rochester, at the home of her brother, F. A. Booker, at Quincy. Mr. Granger, later Judge Granger, as mentioned earlier, was a native of Olmsted County and a brother of Dr. Charles T. Granger; he first was married to Ophelia Cook, a native of Rochester, on June 24, 1896. Mrs. Granger died on April 5, 1898, leaving an infant daughter, Ophelia C. Granger.

After her marriage Dr. Booker Granger managed the dignified home on Third Street, S.W., and carried on her professional work. She was on the staff of the Drs. Mayo until March 1, 1914, when she became a full time health officer in the city of Rochester, again as assistant to Dr. Charles H. Mayo, who since May 1, 1912, had been city health officer. She resigned from public health work after two years to conduct a limited practice as consulting refractionist, with an office on Broadway. In this work she continued until shortly before her death, which occurred at Rochester on July 5, 1928. She was survived by her husband and by relatives in Olmsted County and elsewhere.

Dr. Booker Granger was a member of the Southern Minnesota Medical Association, the Minnesota State Medical Association and the Alumni Association of the Mayo Foundation. A respected citizen of Olmsted County all her life, she possessed the esteem of associates and the many who profited by her skill, and the high regard of friends who were privileged to know her well.

G. W. Green, an eclectic physician, a native of New York born in 1827, came to Minnesota in 1856 because of "a disposition to pulmonary complaint," and settled in Pleasant Grove, Pleasant Grove Township, Olmsted County. He regained his health and, like many of his contemporaries, in addition to practicing medicine ran a general store and engaged in public service. For the term of 1859-1861 he was a member of the Minnesota House of Representatives.

In September, 1861, Dr. Green removed with his wife and four children to Lake City, Wabasha County, where he developed a good practice. It is said that he was briefly in Goodhue County prior to going to Lake City. In 1871 a heavy cold resulted in his prolonged illness, and the summer and autumn of 1872 he spent in California in the hope of improvement. He died on Thanksgiving Day, November 28, 1872, in Lake City. The funeral services were conducted at the Methodist Church, of which he was a member, under the auspices of the local Masonic Lodge.

Stewart V. Groesbeck (1841-1908), an eclectic physician, a native of Otselic, Chenango County, New York, was in High Forest, Olmsted County, the greater part of the time from 1868 into 1872. Earlier, for about a year and a half, into 1865, he had been in Houston County, associated with Dr. P. T. Bowen, who at one time was in Houston. From High Forest Dr. Groesbeck went to Marshall, Minnesota, and thence, after about fourteen years as a citizen and practitioner of some success and importance, he went to Dakota Territory. On June 23, 1887, he received his license to practice medicine in Dakota, having passed the official examination. He was then living in Watertown; by 1906 he was in Spearfish. Later he became surgeon to the National Home for Disabled Volunteer Soldiers at Hot Springs, South Dakota, and he died in that institution on December 31, 1908. He was at the time of his death a member of the American Medical Association.

Biographical notes on Dr. Groesbeck appeared in an article by Eckman, in 1941, on homeopathic and eclectic medicine in Minnesota, and in a paper by Guthrey, in 1945, on the history of medicine in Houston County. Eckman, citing Neill, stated that Dr. Groesbeck in 1871 received a license to practice medicine in Minnesota, and inferred that the doctor had been affected by the Medical Practice Act of March 4, 1869 (soon afterward repealed). Recent information has confirmed this inference: When in May, 1869, the late Dr. David Sturges Fairchild (1847-1930) of Clinton, Iowa, arrived in High Forest with the ink scarcely dry on his diploma from the Albany Medical College, of New York, he found there two physicians, of whom one was Dr. Alexander Grant, well qualified but inactive professionally. The other was an "army doctor," bluff and jovial and popular in the community, who did not possess a record of formal medical study, almost certainly Dr. Groesbeck. Long afterward Dr. Fairchild wrote, in part: "The Medical Practice Act . . . which had created so many vacant places by suppressing the uneducated doctor, was repealed and brought back my predecessor. The doctor . . . who was only theoretically barred from practice, remained in the background and could easily be found. It was humiliating to see a man with no medical training get the patients, but I could only wait." Dr. Fairchild remembered this practitioner as a vender of homeopathic remedies.

(To be continued in the November issue.)

President's Letter

MEDICAL EMERGENCY: WORLD SIZE

With the Korean conflict has come our second world medical emergency in ten years—an emergency of indeterminable length and intensity. What we learned, in the process of discharging our World War II responsibilities, is valuable in assisting us to assume our obligations during this critical period; but the organizational blueprint falls short of 1950 requirements.

As in World War II, a Committee on Procurement and Assignment has been organized within the Minnesota State Medical Association. The committee will work with representatives of the military service in obtaining the necessary number of physicians for duty with the armed forces without inequitable hardship to the communities they serve.

Medical reservists are being summoned by direct call; and the new amendment to the Selective Service Act provides for the induction of men needed in medical, dental and specialist categories. These men, under fifty years of age, will be called initially from the group of ASTP and V-12 students and others deferred to continue their education who have had less than 90 days active service.

But the role of the profession will assuredly not be confined to war service, vital as that duty is. The threat of atomic bombing demands that we become proficient in the care of radiation victims so that a war in our own backyards does not find us hopelessly vulnerable.

Difficult and challenging days lie ahead of us, as we strive to do our part in the prosecution of war without losing our perspective on the continuing problems of America's health.

Nor should we be led, blindly, into a patriotic acceptance of controls that are inconsistent with the freedoms for which the nation is fighting. Certain controls may be necessary, of course, but Americans have built a great nation on the basis of individual responsibility and this same quality of responsibility should be able to carry us through the exigencies of war.

President, Minnesota State Medical Association

Editorial

CARL B. DRAKE, M.D., *Editor;* GEORGE EARL, M.D., HENRY L. ULRICH, M.D., *Associate Editors*

CIVIL DEFENSE

IT IS to be hoped that no atom bomb will ever be dropped on any of our cities. We must admit, however, that the greatest danger facing our country at this moment is a concerted attack on our large centers of population by Russian bombers. Incidentally, the airplane is not the only way an atom bomb could gain entrance to the country. Is there anything to prevent the importation of the component parts of an atom bomb through the Russian Embassy in Washington and its assembling at vital points? There is also no reason for expecting any such warning as a declaration of war as a prelude to atom bombing. As an indication that the Russians have bombing our country in mind is the fact that they themselves are preparing defenses against bombing of vital points in their own country. It is argued that inasmuch as the Russians must know we would never resort to bombing unless we had bombs dropped first on us, their defense preparations indicate that at sometime they will drop atom bombs on us.

It seems a foregone conclusion that any defense against Russian airplanes bearing atom bombs cannot be 100 per cent effective. Any defense measures must of necessity be on a national scale, and a large air force strategically placed and ever on the alert must be relied upon to minimize the possible damage. Airplane defense is being supplemented by the construction of radar and control stations. The establishment of local Ground Observers Corps to implement radar and the Civil Air Raid Warning System is in process in the U. S. Minnesota has completed organization of 264 posts in the 35 counties involved and is now field checking for a test to be conducted by the Air Force in November.

When a bomb is dropped, defense methods planned in advance by each locality will be called into play. If a bomb were to be dropped today, we can be sure no one would know what to do and panic would result. It is high time each unit of population, such as a municipality, city and state, make civil defense plans in case a bomb is dropped.

Much progress has been made in organizing for civil defense on a state basis. An office for Civil Defense for Minnesota has been established at 1003 Commerce Building, Saint Paul (Telephone Garfield 7356). Colonel E. B. Miller is Director and David Harrison, Assistant Director. This office deals directly with the elected heads of local governments and encourages municipalities throughout the state to create local civil defense planning bodies to meet disasters in time of peace or war. Some 250 Minnesota municipalities have already organized planning groups and are in various stages of planning. No community is too small to plan because of the possibility of being called upon to contribute individuals and teams in inter-community co-operation. Stricken areas must receive outside help. Local planners should take inventory at once of existing resources of major importance to disaster relief, such as public utilities, alternate routes and means of transportation, emergency shelters, feeding and clothing of evacuated persons, medical, nursing and hospital facilities, fire fighting equipment, policing, first aid, sanitation, availability of plans and blueprints of existing water, gas, power and light facilities. The various agencies which will be called upon to perform certain duties following a bomb explosion should be informed in advance in order to avoid confusion. The medical profession will be called upon, along with other groups, to perform certain duties and should be prepared.

On October 8, 1948, Governor Youngdahl appointed a Civil Defense Commission which included Dr. A. J. Chesley, secretary of the Minnesota State Board of Health, as chairman of the State Health Section of the Civil Defense. This section is made up of the secretaries of the Minnesota State Medical, Dental, Nurses, Hospital, and Pharmaceutical Associations, the Minnesota Veterinary Medical Society and the Minnesota Public Health Conference. The state has been divided into nine District Health Sections which are actually the same as our Councillor districts.

Each Councillor of the State Medical Association is chairman of his District Health Section which includes representatives of the dental, nursing, hospital, pharmaceutical, and veterinary professions and the M.P.H. Conference. It is the function of each chairman to keep in close touch with the chairman of the city and county Defense Councils and the Red Cross in his district. The Civil Defense Councils of the municipalities are responsible for the execution of the general orders of the Civil Defense Director, and the Medical Health Officers of the eighty-seven counties of the state are members of the local councils. The duties of the councils include co-ordination of health and defense activities locally.

On August 31 a short course on Civil Defense was arranged by Colonel Miller at the University which was attended by about 150 individuals, including councillors, health officers, sheriffs and fire department chiefs. The program included: Effects of Shock and Blast and Fire Attacks on Structures; Mob Psychology and Mass Hysteria; Defense against Atomic Weapons, Using the Geiger Counter, et cetera.

This is a good beginning, but there is an obvious need for the medical profession to inform itself not only on the treatment of victims of atomic bombing, but to make plans in each locality for not only the treatment of bomb victims but their transportation and hospital care.

Dr. Jan H. Tillisch, chairman of the Committee on Military Affairs of the MSMA, in his report to the Council emphasized the need for self-education of the physicians and urged that each county medical society arrange a meeting on Civil Defense to be addressed by someone who has taken a short course such as that mentioned. He also recommended the sending of articles on the treatment of bombing victims to the members by the State Association.*

In meeting such a catastrophe as one caused by an atom bomb, it is obvious that the American Red Cross is not equipped to handle the situation alone. While it functions well in case of floods, tornadoes, fires, earthquakes and epidemics, the Red Cross is too limited in funds to assume large responsibilities in war disasters. Because of its experience with the handling of disasters, however, it can give valuable assistance. The Red Cross has agreed to assist in the program of civil defense in training in first aid and in home care of the sick and injured, in providing food and clothing and temporary shelter, and in participating in a war-time nation-wide blood program. Truly, such a catastrophe as one caused by an atom bomb would require the co-ordinated efforts of everyone planned in advance.

*The following two publications are recommended to the profession: Medical Aspects of Atomic Weapons, Supt. of Documents, U. S. Government Printing Office, Washington 25, D. C. (Price 10 cents) and Effect of Atomic Weapons, same address, (Price $1.25) issued in September, 1950.

SYMPOSIUM ON HYPERTENSION

THE SYMPOSIUM on Hypertension presented September 18-20, 1950, by the University of Minnesota, with the generous financial support of the Mayo Foundation for Medical Education and Research and the Variety Club of the Northwest, passed expectations as far as interest and attendance were concerned. Presented in honor of Drs. Elexious T. Bell, Benjamin J. Clawson and George E. Fahr, the attendance was so great that the Nicholson Hall Auditorium proved inadequate and the main hall of Northrop Memorial Auditorium had to be utilized. An estimated 400 to 500 physicians were in attendance at each session.

The printed program contained the names of many scientists, including physicians and surgeons who have been interested in the subject of hypertension. Opportunity was given for those not on the program to take part in the discussions, and in this group were many whose names are prominently associated with the problem of hypertension.

The various morning, afternoon and evening sessions were presided over by University professors, some located in Minneapolis and others at the Mayo Foundation in Rochester. Doctors Bell, Clawson and Fahr each presided over a session and also appeared on the program.

The dinner in honor of Doctors Bell, Clawson and Fahr was held on the evening of the last day of the session at the Minneapolis Club. Some 150 physicians attended and paid tribute to the retired physicians. Presided over by Dr. George N. Aagaard, Director of Postgraduate Medical Education at the University of Minnesota, to whose efforts the success of the meeting was largely due, the dinner guests were addressed by Dean Harold S. Diehl, Victor Johnson, head of the Mayo Foundation, Dr. Elexious T. Bell and Arthur W. Anderson, president of the Variety Club of the Northwest.

Fortunately, the material presented at the three-

day meeting will be published by the University. Although there is general agreement that the cause of hypertension is unknown and there is no general agreement as to the treatment of the disease, a symposium of this sort at which frankness typified the remarks of the speakers offered a fine opportunity for the exchange of ideas. The published proceedings will afford the reader present-day opinions on the subject of hypertension in a condensed form.

CORONARY THROMBOSIS IN EARLY LIFE

THERE appears in this issue a report by Drs. Parsons and Heimark on the effect of diet and smoking on the prothrombin blood level of a small number of normal individuals. They report that the prothrombin level is lowered after eating a full mixed meal more than after an all-vegetable meal, and by smoking. The authors are searching for a means of detecting such individuals as may be susceptible to coronary thrombosis at an early age and possibly developing measures for prevention.

The authors present their findings and a suggestion for further study. If their findings are confirmed it would not justify a vegetable diet and abstinence from smoking for everyone. The problem of selecting those most likely to develop coronary trouble would still exist.

A low prothrombin time does not produce coronary thrombosis. This is due to sclerotic changes in the coronary arteries, the cause of which is not known but runs in some families. Just what is inherited is not known. Some have incriminated the handling of cholesterol, but blood levels of cholesterol do not correspond with the ingestion of certain fats. Blood pressures are regulated by reflex nerve impulses. If excessive reflex impulses result in hypertension, what is inherited? And does repeated elevation of blood pressure lead to arteriosclerosis? Should those who show an increased blood pressure on using tobacco refrain from its use because of the danger of developing arteriosclerosis of the coronaries?

The authors believe that the difference in prothrombin levels—that is, the low prothrombin time—is what precipitates the coronary attack. That they have not proven. Do some normal persons have a more marked lowering of their prothrombin time by heavy eating and smoking? If further investigation proves this to be the case, should these individuals be put on an anticoagulant constantly—a rather difficult and costly procedure fraught with some danger? And further, will the maintenance of a normal or high prothrombin time prevent a coronary thrombosis? To advise a patient with evidence of coronary arterial disease to abstain from overeating and tobacco is good advice. Whether individuals likely to develop coronary arteriosclerosis can be identified by their prothrombin-time reactions to eating and smoking remains to be proven. The article is provocative of thought and investigation.

REHABILITATION OF HANDICAPPED CHILDREN

Approximately five to seven out of every 100 children who return to school this fall will have a physical limitation, predicts Dr. Alfred R. Shands, Jr., medical director of the Alfred I. duPont Institute of the Nemours Foundation, Wilmington, Delaware.

In an article published in the August, 1950, issue of *The Crippled Child* Magazine, official publication of the National Society for Crippled Children and Adults, the Easter Seal Agency, Dr. Shands says that few people realize the tremendous number of children with physical limitations. If proper care is not given to these children in their early years, the greater number of them will grow into adult life—handicapped both in mind and body.

As Dr. Shands says, "The great majority of these children must look to the public school system for their education, hence, the importance of every classroom teacher knowing what the problem is and how to meet it."

"If time is taken to analyze the child's defects, and if he is guided accordingly, many times the teacher will undoubtedly be the principal factor in the success or failure of the child's life."

Dr. Shands adds that the classroom teacher who knows which children have physical limitations should find out from the parents what has already been done for them and ask the school nurse and doctor what she can do to aid in the child's rehabilitation.

"The teacher's approach to the child should be one of friendliness, warmth, interest, patience, kindliness, and an honest liking for the child," continues Dr. Shands. "The teacher should understand his handicap, realize what his abilities are, and guide him in the pathway of learning and then see that he has the confidence in himself to successfully carry on in life."

The child must come to the realization that, although life is hard, he must face the world. Dr. Shands points out that the crippled child should be given neither excess sympathy nor pity. As early as possible, he ought to learn to do everything he can for himself, and should not be assisted unless absolutely necessary or ask needless favors of others.

"It is inevitable," concludes Dr. Shands, "that the teacher can be truly effective only if she understands the physical needs of the child and can help him toward the best use of his abilities."

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

AMA GETS REPORT ON BRITISH MEDICAL ASSOCIATION CONFERENCE

David Clayton, AMA's London representative, recently reported that the theme of the British Medical Association annual conference was "the ever-present dissatisfaction and frustration felt by the majority of the nation's general practitioners under the National Health Act." Mr. Clayton noted that sixteen resolutions were passed warning the Labour government that if better treatment in remuneration and a decrease in ever-increasing bureaucratic control were not forthcoming, then British doctors should walk out of the scheme in a body.

Mr. Clayton reports that the conference was sparked by a small but extremely vocal body of doctors, the Fellowship of Freedom in Medicine. This group is working hard to try to recover lost ground. Their main line of attack is to "bring before the British public the more flagrant abuses under the scheme, and to keep on plugging away inside the parent body, the B.M.A."

COMMITTEE STUDIES BRITISH MEDICAL EDUCATION

After making a thorough study of the background and evolution of the National Health Service in Great Britain and its relation to medical education there, a special committee of the American Medical Association has submitted its report. The committee, which did its research during December, 1949 and January, 1950, consisted of deans of American medical schools: Harold S. Diehl, M.D., Minneapolis; Loren R. Chandler, M.D., San Francisco; and Stanley E. Dorst, M.D., Cincinnati.

Speaking of the general practitioner and his role in the education picture, the committee reports:

"Even before the National Health Service Act the lot of the general practitioner in Britain was not too happy, and today it is demoralizing. We do not refer to overwork but rather to the fact that he seems destined to a routine life which does not offer the necessary facilities to practice good medicine and which fails utterly to offer the professional incentives which lead to continued growth of the physician. . . . If we really believe that the general practitioner is the 'backbone of medicine,' then let us prepare him for the most difficult task in medicine. After our experience in England, we are confident that this is the most important job facing the medical schools of the United States during the next decade."

The committee report concludes, saying:

"Finally, we would emphasize again the all-important social, economic and geographic background against which British socialism developed. The National Health Service Act is only one facet of British socialism; the welfare state does not exist except as a part of the whole. Furthermore, conditions in Great Britain are so different from those in the United States that it would be folly to contend that what may be necessary for Britain today should be admirable for transfer to the United States. We, fortunately, have the time that is necessary to evolve an adequate medical service for our people without resorting to the centralization of authority in a welfare state."

Takes Stronger View

Taking a more forceful view of the evils evident in European socialism, Dr. William C. Black, former president of the Association of American Physicians and Surgeons, gives first-hand accounts of the detrimental results of the National Health Service in England:

"Stayed in Kenilworth, England for a couple of days. . . . Found out the proprietor of the hotel was an anesthetist—specialist—and had quit a year ago, not because of pay, but because under the regulations of the National Service Act he was not permitted the freedom of judgment and action essential to the best interests of the patient. . . . Rather than do sub-standard work he just quit and now makes a living from the hotel."

This same doctor told Dr. Black, "This Act had the same effect on me, as a physician, as though I had lost an arm or gone blind or developed some

other physical disability which would make it impossible for me to do my work properly."

LOBBY INVESTIGATIONS BRING ACID COMMENTS

After Oscar Ewing recently bragged of his right to crusade for Truman's compulsory medical insurance plan, the *Chicago Tribune* reminded its readers that "Ewing is the most vigorous propagandist of the day in the cause of political medicine." Said the *Tribune:*

"Ewing proclaimed that 'it was not only his right, but his duty' to propagandize in favor of the Truman compulsory medical insurance plan. In this mission he admitted to having the assistance of sixty-five full-time publicity men, paid by the taxpayer."

Magazine Adds Its Voice

Defending the right of the Committee on Constitutional Government to spend $40,000 a month for printing and distributing John Flynn's *The Road Ahead,* the *Saturday Evening Post* avers that the government is the biggest lobby in the country and spends much more than $40,000 to influence legislation. The *Post* quotes Frank Chodorov in Human Events:

"Peanuts and fiddlesticks. Every hour of the day the agencies of the Government spend more than that monthly average to influence legislation, and the legislation they plug is to lengthen their tenure, increase their appropriations, better their emoluments, strengthen their hold on the public purse. . . . The biggest, more pernicious, most unscrupulous and entirely selfish lobby in Washington is the Government. . . . Its list of contributors includes every man, woman and child in the United States. And yet it has the effrontery to point the finger of scorn at an organization that digs up a piddling $40,000 a month for printing and distribution of literature. . . ."

Suggests a Positive Attack

The *Post* then suggests a more vigorous attack against the aggressions of the Buchanan investigating committee:

"We challenge your authority under the Constitution, but we are not embarrassed or ashamed of what we are doing. John Flynn's *The Road Ahead* is an angry book, with here and there an excess of zeal, but it does expose the socialist nature of the Fair Deal program, an analysis already accepted by the British Labor Party. If there is any restriction in America on the right of persons, including corporations, to challenge the pretensions of a political group in this country, it hasn't yet been put in the form of law. Until freedom of speech has been outlawed by Congress, we intend to support our ideas by whatever authorities seem to us likely to arouse the people to their danger.

"The only way the battle for a free economy will be won is by forthright resistance in terms that can be understood, not exclusively by demurrers and legal responses and applications for injunctions, important as these are. If the fight can be staged in an arena where people can see what punches are being thrown, free enterprise may have a chance. It certainly has none if its protagonists always make themselves look like bad boys talking their way out of something sly and discreditable."

American medicine has certainly not been one of those "bad boys" in its open battle against the infiltration of political medicine and the remaining gamut of socialistic schemes.

JOURNAL QUESTIONS MORE SECURITY

Now that President Truman has signed the bill promising social security to some ten million more persons and boosted benefits to those already under its wing, the Wall Street *Journal* can only ask, "What security?"

The most that a family can draw is the promised $150 a month for life, after all principal wage earners have retired. And, the *Journal* says:

"We can hope, too, that the one hundred and fifty dollar bills will fulfill the real promise to these old people, that it will buy for them what they now dream of. We can hope. But in all candor we must say it is a slim hope.

"Before this new $150-a-month level, the maximum for the same family was $85 a month. Five years, ten years ago $85 a month would have bought more security than the $150 will today. At the very best, the raised maximums do no more than catch up with the cheapening of the dollar. To talk about 'increased benefits' in the new law, as the promisers do, is sheer balderdash.

"This time the ink will hardly dry on the new promises before they begin to fade. A hundred and fifty dollars today won't buy what it would have bought last week. And this same government which promises so much is, at the same time, engaged in the same practices—reckless spending and calculated credit inflation—that have in ten years torn the paper dollar in two."

For Instance—

A short, but pointed editorial also appears in the *Journal,* showing by example, the duplication in the numerous bureaus, agencies, boards and committees which carry on the big business of federal government:

"President Truman will add a 'panel' of three members to the existing Loyalty Review Board. It will not replace the Board, but will have the duty of reexamining

the examination of F.B.I. files by the Board. So the Washington correspondents tell us, on the best anonymous authority they can get. That is to say:

Great fleas have little fleas on their back to bite 'em,
And little fleas have lesser fleas, and so *ad infinitum.*
And the great fleas themselves, in turn, have greater fleas to go on;
While these again have greater still, and greater still, and so on."

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg.
Saint Paul 2, Minnesota

J. F. DuBois, M.D., Secretary

District Court of Ramsey County, Minnesota, Declares Vitamins to Be Drugs and Medicines Sale by Grocers and Food Stores Declared Illegal

Re: Milton Culver doing business as Culver's Food Market vs. John Nelson, John J. Pastenacki, Victor E. Feit, J. Norman French, B. A. Deterling and F. W. Moudry (Minnesota State Board of Pharmacy).

In a test case involving the sale of vitamins in tablet, capsule or liquid form, in grocery stores, meat markets, food stores and other similar places, the Hon. Albin S. Pearson, Judge of the District Court of Ramsey County, Minnesota, on August 7, 1950, made an order denying the motion of the plaintiff in the above entitled action for amended findings of fact, conclusions of law, order for judgment or for a new trial. Judge Pearson granted a stay until October 2, 1950, to permit the plaintiff to appeal from the order, or to avail himself of any other legal remedies that he might have.

Mr. Culver, the plaintiff, operating a retail food store in St. Paul, Minnesota, instituted the action against the members of the Minnesota State Board of Pharmacy to have judicially determined the construction and validity of the Pharmacy Act of 1937. The plaintiff alleged that he brought the action for his own benefit and all other retail food stores of the State of Minnesota, similarly situated. The plaintiff alleged that he had a lawful right to sell, in his food store, articles commonly referred to as food vitamins and food supplement vitamins. It was the claim of the plaintiff that such vitamins were sold in the original labeled packages and were for the purpose of supplementing or fortifying the ordinary or usual diet, and that such vitamins were not sold for medicinal purposes or for the purpose of treating or curing any disease of man. The case was tried before Judge Pearson without a jury commencing May 24, 1948. Among the witnesses for the plaintiff were Dr. Bernard Oser, a chemist of New York City, and Dr. Norman Jolliffe, a physician and surgeon of New York City. Among the witnesses for the Minnesota State Board of Pharmacy were Dr. Russell M. Wilder of the Mayo Clinic, Rochester, Minnesota, Dr. Ole Gisvold, Dr. Ancel Keyes and Dr. Raymond Bieter, all of the University of Minnesota, and Dr. Charles Hensel, a physician of St. Paul. Doctors Wilder, Bieter and Hensel testified that vitamins are drugs and medicines. Following the trial the Court permitted each side to submit written briefs outlining their respective positions and setting forth various citations to sustain their respective claims. After thorough consideration of the matter, Judge Pearson, on April 19, 1950, filed the Court's findings of fact, conclusions of law and order for judgment in favor of the defendants.

In his decision Judge Pearson found that vitamins when prepared in tablets, capsules or liquids composed of pure or concentrated vitamins, natural or synthetic, are drugs within the definition of the term "drug" under the laws of the State of Minnesota. The Court also found that such vitamins when offered for sale in the original boxes are not common household preparations sold for nonmedicinal purposes. The Court also found that such vitamins are not harmless proprietary medicines within the meaning of the laws of the State of Minnesota.

Judge Pearson's decision is of the utmost importance, not only to members of the pharmaceutical profession, but to the medical profession and the public generally. The Pharmacy Board was represented in the case by the Hon. J. A. A. Burnquist, Attorney General of the State of Minnesota, and his Chief Deputy George B. Sjoselius.

Surgical Instruments Found in Possession of Albert Lea Man Arrested on Traffic Charge

State of Minnesota vs. Tracy A. King

Early in the morning of September 13, 1950, Richfield, Minnesota, police arrested Tracy A. King, thirty-five years of age, 524 Park Avenue, Albert Lea, Minnesota, on a charge of careless driving. King was driving an Oldsmobile four-door sedan with Minnesota license plates No. 152-808. In the car police found a doctor's medical bag containing numerous surgical instruments, a stethoscope and various medicinal preparations. A receipt was also found made out to "Dr." King. The Minnesota State Board of Medical Examiners was immediately notified by Sheriff Ed Ryan of Hennepin County, and a lawyer for the Medical Board interviewed King. King admitted that he had been representing himself as a doctor; that he had told conflicting stories of having gone to medical schools in Canada and also in Europe. During the questioning, King stated he was born February 2, 1915, at Winnipeg, Manitoba; that he was a Canadian citizen and entered the United States at Noyes, Minnesota, August 15, 1949. He stated that he was a tile setter by trade and had no medical education. He denied that he had actually treated any patients. King stated that his family name was Stroppa, and that he had lawfully changed his name in court at Winnipeg. The matter has been investigated further at Albert Lea, and it has been ascertained that King has an application on file in the District Court there, to become a citizen of the United States. King claimed that he had the surgical instruments and the medicinal preparations in his possession merely for the purpose of "impressing people."

On September 18, 1950, King pleaded guilty in the Municipal Court of Richfield, to a charge of careless driving and was fined $50.00 by Judge Joseph J. Poitras; the fine was paid. The Minnesota State Board of Medical Exanimers is continuing the investigation, and anyone who has any information in reference to King, or any of his medical activities, is respectfully requested to communicate with the Minnesota State Board of Medical Examiners at 230 Lowry Medical Arts Bldg., Saint Paul, Minnesota.

Modern public health does not prevent death alone. It also prevents disease. For every life preserved by a tuberculosis program, scores of individuals are saved from invalidism. For every life saved from malaria, hundreds of individuals are maintained as active producers in the population.—*Am. J. Pub. Health,* August, 1950.

Minneapolis Surgical Society

Meeting of December 1, 1949

The President, Ernest R. Anderson, M.D., in the Chair

CONTROLLED RESPIRATION IN THORACIC AND UPPER ABDOMINAL OPERATIONS

JOHN H. GIBBON, JR., M.D.

Professor of Surgery, Jefferson Medical College
Philadelphia, Pennsylvania

THE PREVENTION of pain has become the least important part of modern anesthesia. Similarly, muscular relaxation for abdominal operations presents no problem to the modern anesthetist. With the solution of these two difficulties modern anesthesia is properly concerned with avoiding disturbance of pulmonary and cardiac function. The function of the lungs is, briefly, to provide entry to the body of the oxygen which is required for metabolic processes and to furnish exit for the carbon dioxide which accumulates as a result of these same processes. The function of the heart is simply to move the blood through the lungs and then to all parts of the body so that these fundamental gas exchanges which take place in the tissues can be compensated for by the reverse process in the lungs.

In the present surgical era the diaphragm has ceased to divide surgeons into the two air tight compartments, as it does the organs of the body. Many upper abdominal operations can be performed with greater ease through a thoracic, or a combined thoracic-abdominal incision. It thus becomes a concern of most surgeons to be interested in this important aspect of modern anesthesia, i.e. the maintenance of normal cardiorespiratory function during operations.

In the absence of adhesions between the lung and the chest wall, opening of one pleural cavity results in the collapse of the lung on that side to about one third of its former volume. This is due to the contraction of the elastic tissue of the lungs. If this elastic tissue were not present and the enormous filming surface, forty square meters, were maintained by stiff alveolar walls, as in a marine or rubber sponge, most of the disturbances of pulmonary function which I am about to discuss, would not occur. With one pleural cavity widely opened the respiratory movements do not result in a normal exchange of gases between the alveoli and the outside air. In addition the respiratory movements, which under these circumstances are greatly increased in magnitude, produce movement of air back and forth between the two lungs instead of in and out of the trachea. The oxygen content and tension of the alveolar gases rapidly declines while the carbon dioxide content and tension rapidly increases. The anoxemia and acidosis which occur are rapidly fatal.

Two ways have been proposed to overcome the collapse of the lung when the pleural cavity is opened. One is to keep the lung exposed to pressure slightly below atmospheric. The other is to maintain a pressure within the lungs which is slightly above that of the surrounding atmosphere. To accomplish the former, Sauerbruch,[7] around the turn of the century, constructed a large chamber maintained at an air pressure slightly below atmospheric, in which the surgeon and his assistants operated. The patient's head projected outside the chamber through a tightly fitting rubber collar. This ingenious but cumbersome apparatus was not widely adopted because of its obvious drawbacks. To accomplish the second method of maintaining expansion of the lungs a positive pressure cabinet was devised. This consisted of a box in which the patient's head was placed through a snug rubber collar which fitted around the neck. The air pressure in this box was maintained slightly above that of the atmosphere. Samuel Robinson[5,6] of Boston, between 1900 and 1910, demonstrated the efficacy of such a positive pressure cabinet in animal experiments and devised one for use with human patients. A simpler method of maintaining positive pressure by the use of a tightly fitting face mask soon replaced the more cumbersome positive pressure cabinet.

Stimulated by Chevalier Jackson's development of the bronchoscope, intratracheal tubes in human patients have now been widely adapted for maintaining a positive pressure within the lungs during intrathoracic operations. These intratracheal tubes ensure an adequate air way at all times and permit the aspiration of any material that may accumulate in the tracheobronchial tree. The great value of these tubes has become so well established that their advantages do not need to be stressed here. Some anesthetists employ these tubes for positive pressure by placing a tightly fitted face mask over them. Others draw the outside end of the tracheal tube through a rubber diaphragm in the face mask and then connect the tube directly with the anesthetic circuit. The drawback to either of these procedures is that, with the use of positive pressure, air passes down the esophagus and enters the stomach, producing gastric distention which requires decompression by stomach tube during the operation or postoperatively. An inflatable rubber cuff around the outside of the lower end of the tracheal tube was soon developed to obviate this difficulty. Gentle distention of this rubber cuff produces for all practical purposes an air-tight system between the air in the lungs

and that in the anesthesia circuit, without the interposition of a face mask. Such a system is that most commonly employed today for intrathoracic operations. The problem of preventing collapse of the lungs has thus been satisfactorily solved. Adequate ventilation of the lungs still remains a problem.

Even with the lung inflated, it is still necessary for the carbon dioxide, which is given off from the blood in the alveoli, to pass from the alveolar air to the atmospheric air. Similarly it is necessary to provide for constant replacement of oxygen in the alveolar air which is taken up by the blood. Pure physical diffusion of gases in the closed air circuit is not enough. There must be a rhythmic movement of gases back and forth between the rubber bag in the anesthetic circuit and the alveoli in the lungs. With one pleural cavity widely opened the normal action of the muscles of respiration do not produce an adequate exchange of gases between the alveolar air and the external anesthetic circuit. The increase in size of the thoracic cage which occurs with normal inspiration merely results in air passing into the pleural cavity of the opened side of the chest, with relatively small amounts of air entering the lungs through the tracheal tube. Expiration similarly lacks effectiveness.

The lack of effective ventilation was not apparent at first, because by the use of a high percentage of oxygen in the rebreathing circuit it has been possible to maintain normal oxygenation of blood during the course of prolonged intrathoracic operations. Thus, if ether is the anesthetic agent, and oxygen is used instead of room air, the oxygen tension in the alveoli can be more than quadrupled. Under these circumstances ventilation may be greatly reduced without affecting the oxygenation of the blood. The story however is quite different with carbon dioxide. Here the question is not one of supplying a gas to the alveoli, but of removing a gas from them. Only adequate ventilation can achieve such removal of carbon dioxide. Probably the fact that cyanosis can be avoided by using a high concentration of oxygen in a closed rebreathing system with an open thorax has drawn attention away from the inadequate ventilation which occurs under these conditions. Unfortunately an increase in the carbon dioxide tension of the arterial blood does not produce any change in the color of skin and mucous membranes, as does the accumulation of reduced hemoglobin in the blood. That the ventilation is inadequate under these conditions of positive pressure breathing has recently been demonstrated by Beecher.[1] He has shown that while adequate oxygenation of the blood can be maintained in the course of long intrathoracic operations, the carbon dioxide tension in arterial blood and alveolar air can rise to quite alarming heights with a concomitant profound drop in the pH of the arterial blood.

Some realization that the ventilation is inadequate under these circumstances has led anesthetists to assist the respiratory movements by gently squeezing the bag with inspiration. This increases somewhat the exchange between the alveoli and the external anesthetic circuit. However, even in these circumstancs, Beecher states that he is only able to prevent a rise in the carbon dioxide tension in about one third of his patients. The reason probably lies in the fact that expiration is not assisted in any way; in fact, the pressure in the external circuit with positive pressure breathing increases with expiration, thus hindering the passage of gas out of the lungs into the breathing bag. Thus with positive pressure breathing unassisted by compression of the breathing bag by the anesthetist, the pressure in the rubber bag and hence in the external circuit decreases with inspiration and increases with expiration. These fluctuations of pressure directly oppose the movement of air from the alveoli to the external circuit, and vice versa. By compression of the rubber bag one phase is helped, but not the other.

Stephens et al[8] in 1947, advocated the use of curare in intrathoracic operations and a maintenance of normal ventilation by manual compression of the rubber breathing bag in the closed circuit. If the bag be kept partially collapsed under these circumstances there is no resistance to expiration, and inspiration is effected by manual compression of the bag. If the bag be kept tense, however, ventilation again becomes impaired due to the increase in pressure with expiration. Such manual control of respiration is a tiresome and repetitive task for the anesthetist in operations lasting many hours, and interferes with his other occupations, such as controlling the depth of the anesthesia, taking the blood pressure, recording the pulse rate and supervising the adminstration of fluids.

Crafoord[2] in 1939, advocated the use of a mechanical apparatus for compressing the rubber bag by air pressure to provide adequate ventilation. He stressed the importance of avoiding any resistance to expiration. He demonstrated in dogs that the accumulation of carbon dioxide in arterial blood could be avoided by this means. Mautz,[3,4] in this country, has made a similar demonstration in animals and has developed a simpler apparatus for the compression of the bag in the respiratory circuit by air pressure. Even more recently Mautz has developed a method of direct mechanical compression of the rubber bag.

Using curare and both Crafoord's apparatus and Mautz's machine, in a large series of intrathoracic and upper abdominal operations, we have been impressed with the very adequate ventilation obtained, as indicated by a study of the arterial blood gases in a small series of patients. With a few exceptions the marked increase in carbon dioxide tension and fall in pH, reported by Beecher, has been avoided. The advantages of the use of such a mechanical apparatus over manual compression of the rubber bag may be listed as follows:

1. The ventilation is adequate enough to avoid a profound drop in pH and marked rise in carbon dioxide tension in the arterial blood.
2. The ventilation is continuous and can be easily adjusted as to rate and depth.
3. The anesthetist is relieved from a manual test which is better carried out by mechanical means.

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Discussion

DR. RALPH T. KNIGHT: By coincidence, we had chosen the same subject for surgical physiological conference at the University when I learned that Dr. Gibbon was to speak on the subject of the "Physiology of Controlled Respiration." We were, therefore, very fortunate in having Dr. Gibbon discuss the subject with us there this afternoon.

Dr. Gibbon should not apologize for talking on this subject, for these problems of physiology associated with anesthesia must be of deep concern to the surgeon and the more familiar he is with them, the better will be the liaison between the surgeon and the anesthesiologist. I am sure that Dr. Gibbon's visit will further increase the prestige of anesthesiology in this community.

Do you not feel, Dr. Gibbon, that the negative pressure chamber and the positive pressure chamber to which you referred were far less physiologically effective than the intermittent positive pressure as we use it today?

You spoke of the elasticity of the lung creating the problem of ventilation by collapsing the lung when the chest is open. However, this same elasticity helps us in ventilation by expelling the gas each time after we inflate the lung.

You spoke of possible damage to the tracheal mucosa by too much pressure in the inflated cuff on the tracheal tube. We had that happen twice. Dr. Dennis then suggested that we inflate the cuff under control by a water manometer and made the first manometer for us. We now make our own cuffs out of contraceptive condoms. These are large enough in diameter to fill the trachea without stretching the rubber and, therefore, the manometer registers to true air pressure against the tracheal mucosa. We do not use more than 10 mm. mercury or 14 cm. water. However, this pressure in the cuff allows us to expand the lungs up to 10 mm. of mercury pressure with each intermittent expansion. More lung pressure than this blocks the pulmonary capillaries and brings pressure upon the heart, interfering with cardiac output.

Dr. Gibbon has been especially interested in adequate pulmonary ventilation to accomplish adequate elimination of carbon dioxide and to keep the pH of the blood at a normal level. I agree with him completely. It is of special interest to me that he found the Mautz machine with the circle type CO_2 absorber to be the most efficient. We have always believed this to be true.

We believe that a frequent test of the tension of CO_2 in the exhaled gas would help us to regulate the controlled respiration to the proper rate and depth. We have not yet been able to obtain an apparatus which will do this as frequently, quickly, conveniently and accurately as we need. Within a month we are to have a mass spectrometer in the operating room which has been prepared by Dr. Nier in our Physica Department. This will do the job, but the apparatus has cost about $15,000. We hope for a simpler and cheaper method.

Up to now we have depended upon the manual manipulation of the breathing bag to control and augment the respiration. A machine such as Dr. Gibbon has used would be a great physical relief, and we are stimulated to try it to see if it will meet the changing demands of the patient and the situation as well as the manual method. Our surgeons seem to need the lung entirely collapsed much of the time in lung surgery, but inflated often enough to keep it inflatable. In heart surgery, we never cease the manual respiration.

Dr. Gibbon's talk has been an inspiration, and we appreciate it very much.

DR. NATHAN KENNETH JENSEN: I have enjoyed Dr. Gibbon's excellent presentation of this very timely problem. We have all thought of respiration in much too limited a sense, our concern being primarily with pulmonary ventilation in relation to oxygen exchange. Dr. Gibbon's paper this evening graphically illustrates the necessity of also giving serious attention to the requirements of carbon dioxide exchange.

In thoracic surgery, we are frequently harder pressed to meet the requirements of carbon dioxide exchange than of oxygenation. The administration of high oxygen concentration will provide adequate oxygenation with very limited pulmonary ventilation, but there is no way to provide for adequate CO_2 elimination except by maintenance of good pulmonary ventilation.

The problem goes much deeper than this however, as respiration for the tissue cells is dependent upon a bellows, the lungs; a pump, the heart; and a distribution system, the circulation. Failure in any of these results in diminished respiratory exchange with resultant respiratory acidosis and anoxia. The anoxia is easier recognized and we are better equipped to correct it than the hypercarbia. An example is the surgical patient with emphysema and deficient pulmonary circulation. This patient will tax the skill of the most experienced medical anesthetist and still may be in severe respiratory acidosis at the termination of a long surgical procedure.

We have encountered severe diffuse capillary bleeding in several patients with inadequate pulmonary reserve who have slipped into respiratory acidosis after several hours of operating time. I would like to ask Dr. Gibbon if he has noted any tendency in patients known to be in respiratory acidosis to bleed excessively?

DR. CLARENCE DENNIS: In the course of his discussion this afternoon and this evening, Dr. Ralph Knight has pointed out that he felt that sudden emptying of the lungs after full inflation would be a very disturbing thing to a surgeon working inside the chest. Have you noted that the motion of the Crafoord respirator is in fact a source of difficulty in doing surgery with the employment of this machine?

I understand, Dr. Gibbon, that you have done some studies on oxygen saturation with continuous tracings throughout induction and performance of a considerable number of operative procedures. Would it be possible for you to tell us anything of your findings in this regard?

DR. GIBBON (closing): I appreciate very much the remarks of Drs. Knight, Jensen and Dennis. With regard to Dr. Knight's comments, I certainly agree that intermittent positive pressure when the thorax is widely opened is more physiologically effective as regards ventilation than the older negative or positive pressure chambers. It is quite true that the elasticity of the lung helps ventilation by aiding expiration with the thorax open. One should remember, however, that this elastic tension is very slight, being equal to the normal negative intrapleural pressure. Consequently the rubber rebreathing bag should not be kept distended as this will interfere with expiration. Dr. Dennis's manometric control of the air pressure in the inflated cuff of the tracheal tube is an excellent one, and should be widely adopted. If the tension of carbon dioxide in the exhaled gas could be rapidly and simply determined during the course of prolonged intrathoracic operations, it would of course be of great assistance in avoiding respiratory acidosis. I trust

(Continued on Page 1034)

Communication

PARATHION POISONING

To the Editor:

Parathion (O,O-diethyl O-p-nitrophenyl thiophosphate) has proved so highly efficient as an insecticide or pesticide that its importance economically is apparent and its usage is rapidly becoming widespread in agricultural communities. Unfortunately parathion is highly toxic for man as well as for insect life. It may be handled and applied safely if, and only if, stringent precautions are strictly observed. However, in spite of the emphasis placed on the need for such precautions excessive absorption may occur through relaxation of these precautions or through accidental heavy exposure. Physicians may therefore be confronted with cases of poisoning from this compound. The management of acute poisoning by a cholinesterase inhibitor is a medical emergency of a type seldom if ever up to this time encountered in medical practice. The following summary of information now available should therefore be of interest to physicians. From this it will be apparent that in this medical emergency the proper timing and dosage of the antidote atropine may be life saving.

The systemic effects of parathion are qualitatively similar to those of other cholinesterase inhibitors, and to the effects of the acetylcholine analogues (pilocarpine, muscarine, arecoline, mecholyl, doryl). Effects of parathion are interpreted as the result of accumulation of endogenous acetylcholine at synapses of the nervous system. They include giddiness, headache, nausea, vomiting, abdominal cramps, diarrhea, miosis, sweating, salivation, lachrymation, confusion, weakness, and muscular fasciculations. A sense of tightness is felt in the chest as the bronchi constrict and fill with mucus. Fatalities appear to result from constriction and secretions in the bronchi or arrest of the heart. On the other hand, recovery from the acute poisoning is usually complete and uneventful. There has been no evidence of permanent injury in such cases.

Treatment may be effective if atropine grains 1/100 to 1/50 (0.65 to 1.3 milligrams) is given at once and every hour or oftener as needed to keep the patient fully atropinized (mouth dry, pupils dilated). If the lungs have filled before the atropine takes effect, clear the bronchi by postural drainage. Oxygen is then indicated. Morphine is contraindicated. Muscular fatigue and weakness may reach a degree requiring artificial respiration. Following even mild symptoms no additional exposure to parathion or other phosphateesters should be allowed until time for cholinesterase regeneration has been allowed.

Intoxication by parathion or other CE inhibitors is an acute episode of 24 to 48 hours. It is terminated by cholinesterase regeneration and is followed by period of gradually decreasing susceptibility to small exposures. Successive parathion exposures may deplete cholinesterase reserves progressively and create a susceptibility to small doses of tetraethyl pyrophosphate or vice versa. Since CE is regenerated rather slowly in man, patients who have suffered parathion poisoning should not be permitted to experience further possible exposures to this compound until it has been established that CE blood levels have returned to normal. Parathion and other phosphate insecticides are not locally irritating, but they produce local cholinergic effects. There has been no chronic or cumulative action other than that on CE as previously described. Dangerous parathion residues have not been detected on food crops sprayed at the proper stage before harvest.

Very truly yours,
AMERICAN CYANAMID COMPANY
D. O. Hamblin, M.D.
Medical Director

MINNEAPOLIS SURGICAL SOCIETY

(Continued from Page 1033)

that Dr. Knight's efforts to accomplish this will prove fruitful.

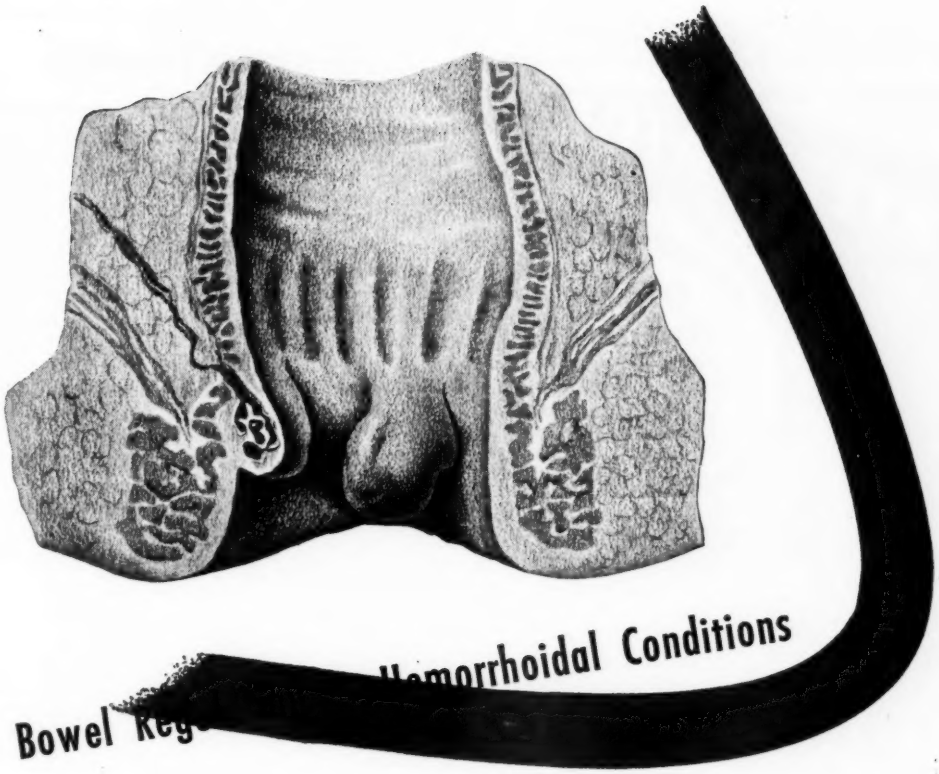
With regard to Dr. Jensen's question concerning profuse capillary bleeding with respiratory acidosis, I might state that we have not made this observation. However, as I stated earlier, we have been able to avoid any serious respiratory acidosis by using mechanical ventilation of the lungs during prolonged intrathoracic operations.

Finally, with regard to Dr. Dennis's questions, I would like to say that we have had no difficulty whatsoever from motion of the lungs using the Crafoord or the Mautz type of mechanical insufflation. With the gentle insufflation pressures used, it is a simple matter to pack the lung out of the way with a moist gauze swab. At intervals during the operation the pressure of the swab is released, and atelectatic portions of the lung are allowed to reexpand. As Dr. Dennis says, we have made some continuous tracings of the oxygen saturation of the blood, using an improved form of the Milliken oxymeter devised by Dr. B. J. Miller of our laboratory. We found little practical use for such continuous recording during the operation but we did learn something, as Dr. Dennis suggests, concerning the induction phase and also concerning the changes which occur in the immediate postoperative period. Because of a momentary period of rather marked unsaturation of blood with oxygen which occurs when sodium pentothal is used to induce anesthesia before introduction of the intratracheal tube, we have discontinued this practice. We now routinely insert the intratracheal tube under local anesthesia, and then with the tube in place we induce anesthesia with a small dose of sodium pentothal continuing with ether and oxygen. Using this procedure we have no period in which the arterial blood is inadequately saturated with oxygen. We have also learned that in the immediate postoperative period, when the patient begins to breath room air, the arterial oxygen saturation tends to decline. This has taught us that it is advisable in patients with poor respiratory reserve to continue the administration of oxygen in the immediate postoperative period until the patient has been returned to his bed and placed in an oxygen tent.

I appreciate the honor of having been invited to speak before the Minneapolis Surgical Society. It has been a great pleasure to be here.

The meeting adjourned.

WILLIAM H. RUCKER, M.D., *Recorder*



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AUXILIARY MEMBERS URGED TO VOTE

Mrs. L. R. Scherer Legislative Chairman

All members of the Woman's Auxiliary to the Minnesota State Medical Association are urged to vote in the November elections. Auxiliary members, as individuals, should support and work for individual candidates.

For information about any candidate, write to Mrs. L. Raymond Scherer, 1930 Irving Avenue South, Minneapolis.

Also of help in keeping Auxiliary members informed will be the radio programs soon to be released by the American Medical Association.

BULLETIN SUBSCRIPTION CALLED IMPORTANT

Mrs. Peter S. Rudie, Bulletin Chairman

The RIGHT and WRONG questions about *The Bulletin* of the Woman's Auxiliary to the American Medical Association:

It is RIGHT to subscribe to *The Bulletin* because:

1. It is to the Auxiliary member what *The Journal* is to the doctor.
2. It contains valuable information on present-day problems of concern to the medical profession and the auxiliaries.
3. It tells of activities of all the auxiliaries in the United States.
4. It is helpful to auxiliaries because it gives new ideas for taking part in public health, health education and public relations activities.
5. Every county officer and auxiliary member should subscribe to *The Bulletin.*

It is wrong:

1. *NOT* to subscribe to *The Bulletin!*

The Bulletin is issued quarterly—August, December, March and May. Subscription price is one dollar a year. Each *Bulletin* Chairman is asking for subscriptions NOW. Names and addresses should be sent in IMMEDIATELY.

PROBLEMS IN TUBERCULOSIS

Even after clinical follow-up in minimal tuberculosis has confirmed the interpretation of the ill-defined x-ray shadow, the physician is faced with another and perhaps more serious problem. He must then cope with the question of the lesion's significance, and must decide upon the course of action to be taken in its management. Will the patient need to undergo hospitalization and surgical procedure? Can the lesion be managed under a home-care regimen? Or will it be sufficient to place the patient under long-term observation, imposing only token limitations upon normal activity? It will be most urgent that these questions be resolved properly and decisively.

These are but a few of the problems which our screening survey experiences in communities and hospitals pose for us and for the medical profession generally. Meeting them directly and fully is the best assurance of effective tuberculosis control.—ROBERT J. ANDERSON, M.D., *Journal-Lancet,* April, 1950.

In Memoriam

CHARLES ANTHONY REED

Dr. Charles A. Reed, well known orthopedist of Minneapolis, died August 22, 1950, at Eitel Hospital. He was seventy-eight years of age.

Dr. Reed was born February 2, 1872, at Hastings, Minnesota. He received his medical degree from the University of Minnesota Medical School in 1898. He first practiced medicine at Kalispell, Montana, and in 1903 and 1904 studied orthopedic surgery in Germany. He began practicing medicine in Minneapolis in 1909. In 1920, he helped organize the Nicollet Clinic and became associated with the staff of Eitel Hospital. He held an appointment as associate clinical professor of orthopedic surgery at the University of Minnesota Medical School and was assistant chief surgeon at the Shriner's Hospital for Crippled Children in Minneapolis.

During World War I, Dr. Reed served as a major with Base Hospital 26 in France. He was a member of the American College of Surgeons and a fellow of the American Academy of Orthopedic Surgeons. He was a member of the Hennepin County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Dr. Reed's wife, the former June Clarke Dickey of Minneapolis, died in 1947. They were married in 1916. He is survived by a stepdaughter, Mrs. S. B. Marantz.

MARK E. RYAN

Dr. Mark E. Ryan of Saint Paul, Minnesota, died following a coronary attack on August 16, 1950. He was sixty years of age.

Dr. Ryan was born at Delafield, Wisconsin, May 4, 1890. He attended school at Oconomowoc, Wisconsin, and obtained a B.S. degree from the University of Wisconsin in 1918. He attended medical school at the University of Wisconsin and at the University of Minnesota, graduating from the latter in 1921. He interned at [illegible] land General Hospital in 1921 and began practice in Saint Paul with his brother, Dr. John J. Ryan, in 19[illegible].

Dr. Ryan was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association. He had been a member of the Ancker Hospital staff for many years and had been active in medical, religious and civic circles in Saint Paul.

Dr. Ryan is survived by his wife, Isabel Perry Ryan; four sons, Mark E., James P., Lawrence P., and Paul W.; a daughter, Sister Mary of St. Mark of the House of Good Shepherd; two brothers, Dr. John J., of Saint Paul, and Dr. William, of Duluth.

The continued responsibility for the care of a chronically sick person adds immeasurably to the education of a physician. It requires maturity to be able to recognize limitations, to avoid becoming angry because the patient does not get well, to avoid becoming discouraged or discouraging, and to continue to wish to help within the limits of one's ability.—JOHN ROMANO, M.D., *J.A.M.A.*, June 3, 1950.

✦ Reports and Announcements ✦

OMAHA MID-WEST CLINICAL SOCIETY

The Omaha Mid-West Clinical Society will hold its eighteenth annual assembly at Hotel Paxton, Omaha, Nebraska, October 23 to 27, inclusive.

The general program plan for 1950 will be much the same as in previous years. Distinguished guest speakers who are eminent in their particular specialty fields will present addresses, clinics and question-and-answer periods; members of the society will present lectures, panel discussions and scientific exhibits; a guest panel on the antibiotics has been scheduled for Friday morning. Scientific motion pictures will open the daily program, and technical exhibits will again be on display.

The annual sessions of the Omaha Mid-West Clinical Society have received a Class A rating from the American Academy of General Practice. This means that Academy members who attend the sessions will receive credit toward the fifty hours of *formal* postgraduate study required of them every three years.

Further information may be obtained by writing to the executive office of the Society, 1031 Medical Arts Building, Omaha, Nebraska.

INSTITUTE OF INDUSTRIAL HEALTH

The Institute of Industrial Health of the University of Cincinnati will accept applications for a limited number of fellowships which are being offered to qualified candidates who wish to pursue a graduate course of instruction which will qualify them for the practice of industrial medicine. Candidates who complete satisfactorily the course of study will be awarded the degree Doctor of Industrial Medicine. Any registered physician, who is a graduate of a Class A medical school and who has completed satisfactorily two years of residency (including internship) in a hospital accredited by the American Medical Association may apply for a fellowship in the Institute of Industrial Health. The course of instruction consists of a two-year period of intense preliminary training in the basic phases of industrial medicine followed by one year of practical experience under adequate supervision in industry. During the first two years, the stipends for the fellowships vary from $2,100 to $3,000. In the third year the candidate will be compensated for his service by the industry in which he is completing his training. Requests for additional information should be addressed to the Institute of Industrial Medicine, College of Medicine, Cincinnati 19, Ohio.

UROLOGY AWARD

The American Urological Association offers an annual award of $1000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some

(Continued on Page 1042)

UROLOGY AWARD

(Continued from Page 1040)

clinical or laboratory research in urology. Competition shall be limited to urologists who have been in such specific practice for not more than five years and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Palmer House, Chicago, Illinois, May 21 to 24, 1951.

For full particulars write the secretary, Dr. Charles H. de T. Shivers, Boardwalk National Arcade Building, Atlantic City, New Jersey. Essays must be in his hands before February 10, 1951.

POSTGRADUATE CONFERENCE IN OTOLARYNGOLOGY

The annual Postgraduate Conference in Otolaryngology at the State University of Iowa, will be conducted November 27 to December 2 at the University Hospitals, Iowa City, Iowa. Further information may be obtained from the Director of Medical Postgraduate Studies, Medical Laboratory Building, Iowa City, Iowa.

RADIOLOGICAL SOCIETY OF NORTH AMERICA

The Radiological Society of North America will hold its thirty-sixth annual meeting in Chicago, December 10 through 15. Headquarters will be at the Palmer House where scientific and technical exhibits and sessions will be held. All members of the profession are welcome and are invited to attend.

CONTINUATION COURSES

Diseases of the Chest.—A continuation course for physicians in diseases of the chest will be presented at the University of Minnesota Center for Continuation Study, October 26 to 28. The course is intended for general physicians and is presented with the sponsorship and financial support of the Minnesota Trudeau Society. Distinguished visiting physicians who will participate as faculty members of the course include Dr. O. A. Sander, associate in medicine, Marquette University Medical School; Dr. John H. Skavlem, president of the American Trudeau Society, and associate professor of medicine, Cincinnati University Medical School; and Dr. James J. Waring, professor and chief of medicine, University of Colorado. The remainder of the faculty for the course will be made up of members of the staff of the University of Minnesota, the Mayo Foundation, and the Minnesota Trudeau Society.

Child Psychiatry.—A continuation course in child psychiatry for pediatricians and physicians will be presented at the Center for Continuation Study, November 27 to December 1. Dr. Ralph D. Rabinovitch and Dr. John Waterman will participate as lecturers and group discus-

(Continued on Page 1044)

CONTINUATION COURSES

(Continued from Page 1042)

sion leaders. Dr. Reynold A. Jensen, head of the Child Psychiatry Service of the University of Minnesota, is in charge of the arrangements for the course and will also act as lecturer and group discussion leader.

Poliomyelitis.—A continuation course on poliomyelitis will be presented at the University of Minnesota Center for Continuation Study on November 9 to 11, with the sponsorship of the Elizabeth Kenny Institute. The course is intended for doctors of medicine engaged in general practice and for such specialists as pediatricians, physiatrists, orthopedic surgeons, and neurologists. Dr. Harold A. Sofield, associate professor of the Department of Bone and Joint Surgery, Northwestern University Medical School, will be the visiting faculty member for the course.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

Approximately fifty physicians attended the two-day annual meeting of the Northern Minnesota Medical Association in Bemidji on September 8 and 9. The meeting was held under the direction of Dr. W. J. DeWeese, president of the group during the past year.

Officers elected during the business session include the following: Dr. L. W. Johnsrud, Chisholm, president; Dr. G. A. Sather, Fosston, re-elected vice president; Dr. C. L. Oppegaard, Crookston, re-elected secretary-treasurer.

SOUTHERN MINNESOTA MEDICAL ASSOCIATION

At the annual meeting of the Southern Minnesota Medical Association in Mankato on September 11, Dr. R. F. Hedin, Red Wing, was elected president of the organization. He succeeds Dr. Warren E. Wilson of Northfield.

Other officers named at the meeting are Dr. C. W. Rumpf, Faribault, first vice president; Dr. H. G. Nilson, Mankato, second vice president, and Dr. W. A. Merritt, Rochester, secretary-treasurer.

The 1951 meeting of the association will be held in Rochester.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The Minnesota Society of Neurology and Psychiatry held its regular meeting in Saint Paul on September 12. Dr. J. C. Michael, Minneapolis, presented a discussion of "Antabus Therapy in Alcoholism," and Dr. Harold F. Buchstein, Minneapolis, spoke on "Prefrontal Lobotomy in the Relief of Pain."

PENNINGTON COUNTY SOCIETY

In the first election of officers conducted by the recently organized Pennington County Medical Society, at a meeting in Thief River Falls on August 23, Dr. O. F. Mellby was elected president of the group. Other officers elected include Dr. Harold C. Johnson, vice president, and Dr. George T. Van Rooy, secretary-treasurer. All are of Thief River Falls.

(Continued on Page 1046)

During the scientific part of the meeting Dr. A. E. Culmer of Grand Forks, North Dakota, presented a paper on the operative treatment of fractures.

RED RIVER VALLEY SOCIETY

The Red River Valley Medical Society held a meeting in Crookston on July 22. Principal speaker at the afternoon session was Dr. Richard E. Reiley, Minneapolis, who discussed fractures and fracture problems as the orthopedist sees them referred by the general practitioner.

A feature of the evening session was a discussion of the Minnesota medical educational campaign by Lyle A. Limond, field secretary of the Minnesota State Medical Association.

WASHINGTON COUNTY SOCIETY

At the regular monthly meeting of the Washington County Medical Society held at Stillwater in September, Dr. Emmerson Ward of Rochester spoke on "Cortisone in Therapeutics." Visitors attending the meeting included Dr. D. A. Burlingame, Saint Paul, Dr. Campbell of New Richmond, Dr. Cornwall of Andover, and Dr. Bourget of Hudson, Wisconsin.

CEREBRAL PALSY CLINIC

The Cerebral Palsy Council, Inc., of Minnesota, 279 Rice Street, Saint Paul, is endeavoring to develop a cerebral palsy registry. Its purpose is to acquaint families of these persons with groups in various vicinities who are studying the problem. Registry cards were sent out last spring to secretaries of all county medical societies. Secretaries are urged to return these cards with necessary information on each as quickly as possible.

SALT WATER ORALLY FOR SHOCK

A group of leading American surgeons has advised the Public Health Service, Federal Security Agency, that salt water taken by mouth, in a vast majority of cases, is as effective as blood plasma in the emergency treatment of shock from serious burns and other injuries.

The recommending surgeons are members of the Surgery Study Section, an advisory body to the National Institutes of Health and to the Surgeon General of the Public Health Service.

In general terms, the treatment calls for approximately one level teaspoonful of table salt and one-half teaspoonful of baking soda for each quart of water. A number of quarts are required each day. The only limitations on the amount consumed is the ability of the patient to consume the saline solution. Since great thirst accompanies serious burn injury, it has been found that patients will voluntarily consume a sufficient amount of the solution, which is quite palatable. No other drinking fluid is permitted in the first few days following injury.

In releasing the recommendation, Surgeon General Leonard A. Scheele said:

"Salt water offers an easy, practical method for the treatment of shock which follows serious burns and other injuries. It is particularly important in any period of large scale disaster. Unless the patient is disoriented, is in acute collapse or is among the very small percentage who become nauseated by drinking large quantities of the salt solution, the sodium chloride formula will be effective when administered by mouth."

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Of General Interest

Dr. John F. Pohl, Minneapolis orthopedic surgeon, is the author of a recently published book entitled "Cerebral Palsy." The book, which is said to be the first complete medical text on cerebral palsy, was published by the Bruce Publishing Company, Saint Paul and Minneapolis.

* * *

Dr. Titus C. Kreuzer, of Marshall, who recently returned from a several months' tour of Europe, writes that there was a considerable difference of opinion in England concerning the national medical service. "It was very definite," he writes, "that the working class seemed to be in favor of the 'free medical care,' as they called it, because it didn't cost them anything. However, the white-collar class was very bitter about the plan, stating that they had always been able to care for their own needs and could continue to do so. They said they did not want to get into the queue but wanted service when they needed it."

Dr. Kreuzer adds that "in Germany, Italy and France it was very evident that a preponderance of the lay people, as well as physicians, wanted to get to America if possible."

* * *

Dr. Conrad I. Karleen, formerly associated with Dr. Carl W. Waldron of Minneapolis, has announced the removal of his office to 402 Medical Arts Building, Minneapolis. His practice is limited to plastic and reconstructive surgery.

* * *

Dr. William H. Inglis has joined the staff of the Dr. R. J. Cairns clinic in Redwood Falls. Dr. Inglis was graduated from the University of Minnesota Medical School in 1949.

* * *

Red Lake Falls acquired a new physician in August when **Dr. Allan McKaig** arrived from Birmingham, Alabama, to be associated in practice with Dr. Lester N. Dale. A graduate of the University of Syracuse, Dr. McKaig spent two years in the armed services and then practiced at Birmingham for two years. Dr. McKaig and Dr. Dale will practice in newly remodeled offices in a building which Dr. Dale purchased in August.

* * *

Dr. Kenath Herrick Sponsel has moved his offices to 321 Medical Arts Building, Minneapolis, to continue the practice of the late Dr. Vernon L. Hart. Dr. Sponsel's practice will be limited to orthopedic and traumatic surgery.

* * *

Dr. Valentine O'Malley has opened offices for the practice of internal medicine at 541 Lowry Medical Arts Building, Saint Paul.

Since 1948 the **American Diabetes Association** has conducted and financed a yearly detection drive through its special Diabetes Detection Committee. It is estimated that there are a million unknown cases of diabetes in the country. Early detection means better control and more normal lives for those so afflicted. Last year about 7,500 unknown diabetics were uncovered during Diabetes Week. It is expected that Diabetes Week this year, scheduled for November 12 through 18, will be even more successful.

The American Diabetes Association has prepared a special article designed for use in company publications and those of labor organizations, with the aim of facilitating acceptance of the campaign by employes. Copies of the article may be obtained from the office of the American Diabetes Association, 11 West 42nd Street, New York, N. Y.

The campaign is a program of the medical profession, approved by the AMA, and needs the support of the community. Last year, through the cooperation of the local county medical society and the Chamber of Commerce in Virginia, Minnesota, 9,791 residents of the city were tested and at least fifty new cases of diabetes were discovered.

* * *

Dr. Gordon R. Kamman, Saint Paul, spoke on "Psychosomatic Medicine" at the monthly meeting of the Lyon-Lincoln Medical Society at Marshall on September 19.

* * *

Dr. Harry W. Christianson and **Dr. Robert J. Tenner,** Minneapolis, have announced their association with Dr. Lloyd F. Sherman, who was formerly associated with Dr. Harry E. Bacon of Philadelphia in the practice of proctology.

* * *

The American Dermatological Association is offering a prize of $300 for the best essay submitted on original work not previously published relative to some fundamental aspect of dermatology or syphilology. Manuscripts, double spaced, should be submitted in triplicate not later than February 1, 1951, to Dr. Louis A. Brunsting, Secretary, American Dermatological Association, 102 Second Avenue S.W., Rochester, Minnesota.

* * *

Dr. John S. Hamlon has become a member of the staff of the state hospital at Fergus Falls. A graduate of the University of Minnesota Medical School, Dr. Hamlon served for five years in the Army in Africa and Europe. Following his release from the Army he practiced at St. Charles until his move to Fergus Falls in August.

(Continued on Page 1050)

It was announced on August 24 that **Dr. Joseph C. Belshe** planned to leave Northfield by September 15 to become associated in practice with Dr. Fred B. Riegel at St. Croix Falls, Wisconsin. Dr. Belshe has been associated in practice in Northfield with Dr. Robert F. Mears.

* * *

In August the Clearwater Clinic of Bagley purchased a clinic building at Gonvick and announced that a physician would be secured to replace **Dr. Norman F. Stone,** of Gonvick, who had received orders to report for duty in the Navy. It was expected that Dr. Stone would leave Gonvick about November 1.

* * *

Dr. G. T. Schimelpfenig and **Dr. B. H. Simons,** Chaska, attended a meeting in Shakopee on August 24 at which plans for a tri-county community hospital project were discussed.

* * *

The engagement of **Dr. Jack Gordon Olsen,** formerly of Edina, and Miss Nancy Elizabeth Harris, Durham, North Carolina, was announced on August 27. The wedding is scheduled for October 27. Dr. Olsen is a graduate of the University of Minnesota Medical School.

* * *

The following statement on prophylaxis against subacute bacterial endocarditis was approved at the annual meeting of the **American Council on Rheumatic Fever** on June 12:

Following dental extractions and removal of tonsils and adenoids, bacteria are frequently present in the blood stream for short periods of time. In rheumatic individuals or in patients with congenital heart disease these bacteria may lodge in the heart valves and cause bacterial endocarditis. Although a variety of bacteria cause this disease, the majority of cases are due to alpha streptococci (Streptococcus viridans). Alpha streptococci are usually resistant to sulfa drugs. Penicillin is, therefore, recommended for prophylaxis.

1. Except in emergencies, operative procedures in rheumatic individuals should be deferred until there is no clinical evidence of rheumatic activity and laboratory tests indicate that the rheumatic process is subsiding.

2. Patients should be free of upper respiratory infection.

3. Minimum dosage of penicillin: (a) 300,000 units of aqueous penicillin injected intramuscularly thirty to sixty minutes before extraction or operation; (b) 300 000 units of procaine penicillin in oil injected intramuscularly at the same time in a different site.

Penicillin prophylaxis is not necessary for the extraction of deciduous incisors or bicuspids unless infection of the gum is present. It should be used for the extractions of deciduous molars, all permanent teeth and for tonsillectomy and adenoidectomy. In most instances it is best to extract one tooth at a time; multiple extractions should be avoided. In cases of extensive gum infection or severe root infections (apical abscesses) it is advisable to give several doses of penicillin, starting the day before

(Continued on Page 1052)

(Continued from Page 1050)

operation and continuing one or two days thereafter.

Women with rheumatic or congenital heart disease should receive penicillin prophylaxis at the time of delivery. It is also recommended for patients requiring gastrointestinal surgery.

* * *

Dr. J. J. Ahlfs, Caledonia, has announced that **Dr. Hildegard J. Virnig,** formerly of Mount Morris, Illinois, has become associated in practice with him. A graduate of the University of Minnesota, Dr. Virnig has done postgraduate work in pediatrics and gynecology.

* * *

A chest clinic was conducted in Austin on August 28 by **Dr. Karl H. Pfuetze,** director of Mineral Springs Sanatorium, Cannon Falls.

* * *

At a meeting of the South Dakota Medical Society in Spearfish, South Dakota, during the middle of August, **Dr. William H. Bickel,** Rochester, presented a paper entitled "Acute Fracture Complications."

* * *

Dr. and Mrs. L. R. Parson, Elbow Lake, were honored on the occasion of their thirtieth wedding anniversary at a dinner party given by friends at Sandy Point on August 31.

* * *

Dr. Roger F. Hartwich, who has practiced in Winona for more than a year, joined the staff of the Winona Clinic on September 5.

* * *

Dr. J. Arthur Myers, professor of public health and preventive medicine at the University of Minnesota, left for Rome on September 12 to speak at the first International Congress on Diseases of the Chest. He also was scheduled to lecture at the Hebrew Medical School in Jerusalem.

* * *

Dr. Malcolm J. Lester, formerly of Fairmont, has taken over the general practice of Dr. C. F. Medlin in Truman. Ill health forced Dr. Medlin to retire from active practice.

Dr. Lester, a graduate of the University of Louisville, interned at Swedish Hospital, Minneapolis, then was associated in practice with Dr. Harold Coulter in Madelia. Before moving to Truman he was practicing with Dr. R. S. Hunt and Dr. R. C. Hunt in Fairmont.

* * *

After practicing in Jackson for a year, **Dr. Curtis M. Johnson** left during the first week of September to begin practice elsewhere. Before leaving Jackson, Dr. Johnson stated that his plans were somewhat uncertain because of the changing military situation.

* * *

The offices of **Dr. F. W. Behmler** and **Dr. R. A. Rossberg** in Morris have been completely remodeled and enlarged. The changes were made to provide accommodations for Dr. J. C. Kooda, who, it was

(Continued on Page 1054)

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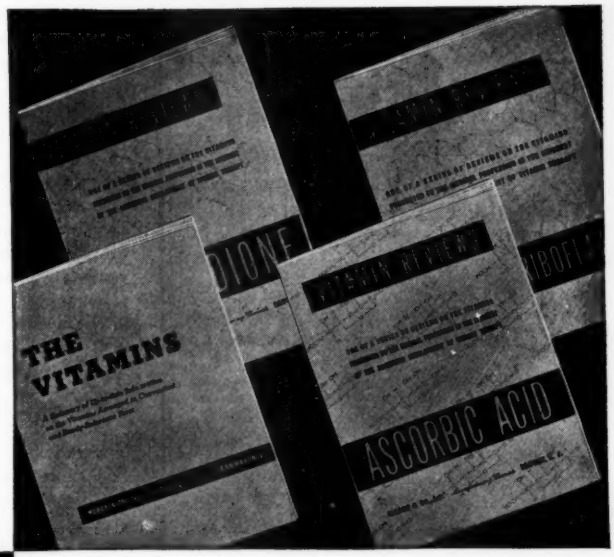

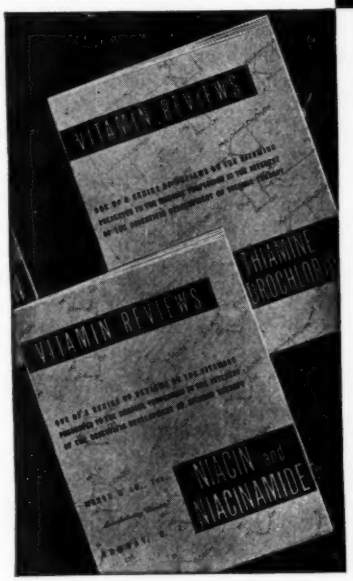

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- Distribution in foods.
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- Clinical use in specific conditions.

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(Continued from Page 1052)

announced, was moving from Eagle Bend to practice in Morris.

* * *

It was announced on August 31 that **Dr. Harold J. Stoen** was planning to open offices for the practice of medicine in Anoka about September 18. A graduate of Rush Medical School in 1934, Dr. Stoen interned at Fresno General Hospital, California, and then completed a one-year residency in surgery at the Home Hospital, Lafayette, Indiana. After three years as a staff member of the U. S. Marine Hospital at Cleveland, Ohio, he began general practice in Lafayette in 1940. With the exception of four years with the Army Air Force during World War II, he practiced continually at Lafayette until 1950.

* * *

Dr. Robert Nash Evert and Miss Doreen Alma Nerlund were married on September 29 in the Como Park Lutheran Church, Saint Paul. Dr. John Evert, the brother of the groom, was best man. The bridegroom is the son of Mrs. John Evert and the late Dr. John Evert of Glendive, Montana.

* * *

Dr. Lawrence J. Swanson has opened offices for the practice of medicine at 950 South Robert Street, West Saint Paul. Dr. Swanson was graduated from the University of Minnesota Medical School in 1946.

* * *

Dr. Alvin Erickson of the Long Prairie Clinic spent two weeks in September at the Cook County Hospital, Chicago, taking postgraduate courses.

* * *

Dr. F. R. Heilman and **Dr. W. E. Herrell,** Rochester, have been named to the editorial board of a new medical journal, "Antibiotics," which will make its first appearance in January. Dr. Heilman is with the bacteriology division of the Mayo Clinic, and Dr. Herrell is with the diagnostic divisions of general medicine and surgery.

* * *

Dr. Frank J. Hill, Minneapolis health commissioner, has been named to the founders group of the American Board of Preventive Medicine and Public Health.

* * *

Dr. Mark Anderson, Jr., Rochester, a fellow in surgery in the Mayo Foundation, reported for active duty with the Navy on September 5. He was the first fellow from the foundation to be called into active service.

* * *

Dr. Evelyn E. Hartman, formerly with the department of baby clinics in Finland, has joined the staff of the Minneapolis city health department to serve as maternal and child health physician.

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Dr. Howard Kaliher left Pelican Rapids late in September to move to Tillamook, Oregon, where he had accepted an appointment to the staff of the Tillamook County Hospital.

Among the speakers at the annual meeting of the Idaho State Medical Association at Sun Valley, Idaho, during the first week of September were **Dr. John S. Lundy** and **Dr. Robert Kierland.**

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Dr. Mitrofan Smorszczok, Polish displaced person who was granted his medical license in Minnesota this summer, began practice in Monticello in mid-August. He is associated with Dr. William E. Hart of Monticello. It was Dr. Smorszczok's arrival in Minnesota in 1948 that started a controversy about the state rules barring foreign-trained physicians from licensure.

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Dr. Leonard M. Ellertson, a graduate of the University of Iowa Medical School, has become associated in practice with Dr. C. E. J. Nelson and Dr. O. A. E. Erdal in Albert Lea. Dr. Ellertson has served in the Navy and has been a resident physician at the Ball Memorial Hospital, Muncie, Indiana.

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Dr. and Mrs. J. F. Weir, Rochester, left during the first week of September for a two-month trip to Europe. In addition to sightseeing, Dr. Weir, who is head of a section in medicine at the Mayo Clinic, attended the First International Congress of Internal Medicine at the University of Paris during the middle of September.

Dr. Hector M. Brown, medical director of the Walker Hospital at Walker, has opened a branch office in Backus for the practice of medicine. The office is open on Saturday mornings.

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After forty-six years of medical practice, forty-two of which were spent in Northfield, **Dr. I. F. Seeley** retired from active practice and moved to Tucson, Arizona, on August 27.

On the day of departure a reception was held at the Northfield Masonic Hall in Dr. Seeley's honor. Representatives from surrounding cities were present to pay tribute to Dr. Seeley for his years of service. A few days earlier, on August 24, Dr. and Mrs. Seeley were honored at a dinner given by Northfield physicians and the Northfield Hospital board and nursing staff.

A graduate of the University of Iowa in 1904, Dr. Seeley began his practice in Elysian. After a year there he spent two years as a physician with construction companies building the Milwaukee Railroad through the Rocky Mountains. He settled in Northfield and opened his practice there in 1908. For the past two years Dr. Stanley T. Kucera has been associated in practice with him.

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Dr. J. W. Janes, Rochester, spoke on "Common Bone Malignancies" at a meeting of the Iowa State Medical Society at Carroll, Iowa, on September 7.

Dr. Willis E. Lemon, Rochester, a fellow in radiology in the Mayo Foundation for the past three and one-half years, joined the x-ray department of the Chesapeake and Ohio Railroad Hospital in Clifton Forge, Virginia, on September 12. Dr. Lemon is the son of Dr. and Mrs. Willis S. Lemon of Rochester.

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Dr. Ernest M. Hammes, Jr., Saint Paul, participated in the postgraduate course for general practitioners held at Charles City, Iowa, September 18, through the presentation of a paper entitled "Common Neuroses and Their Management." The course was presented under the auspices of the Iowa State Medical Society.

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Dr. Gordon M. Martin, Rochester, was named fifth vice president of the American Congress of Physical Medicine at the organization's twenty-eighth annual meeting in Boston during the last week of August.

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Dr. Leon L. Adcock and his wife, **Madeline S. Adcock,** have decided to dedicate themselves to the work of medical missions. They will establish a dispensary at Berekum on the Gold Coast of Africa under the direction of the Medical Mission Sisters. Until recently they served as resident physicians at St. Joseph's Hospital, Saint Paul.

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Dr. H. L. Smith, Rochester, presented a paper entitled "The Movements and Sounds of Heart Valves in Various Laboratory Animals" at the International Cardiologic Congress in Paris on September 9.

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The Golden Anniversary Dinner of the *American Journal of Nursing* was held on October 10, 1950, at the Waldorf-Astoria Hotel, New York.

Otto L. Wiese, editor and publisher of *McCall's Magazine,* acted as toastmaster. Serving on the Committee of Sponsors for the occasion were: Helen Hayes; Dr. Elmer L. Henderson; Senators Irving M. Ives and Herbert H. Lehman; Henry R. Luce, editor of *Time, Life* and *Fortune;* General George C. Marshall, and Mary Martin, star of South Pacific. Mrs. Eleanor Roosevelt addressed the guests on the subject, "The Nurse and the World of Tomorrow."

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The Health Insurance Council, made up of the leading trade associations in the life and casualty insurance fields, has reported great gains in voluntary health insurance in 1949.

At the year's end 44 per cent of the entire population was covered by hospital insurance and nearly 60 per cent of the employed civilian population was protected against loss of income because of disability.

The total number of persons covered under voluntary accident and health insurance plans for hospital expense increased in 1949 to 66,044,000 from 60,995,000 a year earlier, a gain of 8 per cent. Those covered for surgical expense increased to 41,143,000 from 34,060,000, an increase of 21 per cent.

Coverage for medical expense increased from 12,895,000 to 16,862,000, a gain of 31 per cent.

The following men trained in the Division of Dermatology at the University of Minnesota passed recent examinations of the American Board of Dermatology and Syphilology: **Robert W. Goltz, Melvin Grais, Stanley Huff, Harold Hurst,** and **Wm. Macauley.** Dr. Henry E. Michelson, Minneapolis, is president of the American Board of Dermatology and Syphilology.

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HOSPITAL NEWS

Officers were elected at staff meetings recently of the following hospitals:

St. Michael's Hospital, Sauk Centre.—Dr. J. F. DuBois, Sr., Sauk Centre, was elected president of the medical staff of St. Michael's Hospital at a meeting on August 29. Other officers include Dr. A. H. Zachman, Melrose, vice president, and Dr. John C. Grant, Sauk Centre, secretary-treasurer. The new hospital, a fifty-bed institution, was formerly opened on September 1.

St. Francis Hospital, Shakopee.— Dr. M. B. Hebeisen, Chaska, was named chief-of-staff of St. Francis Hospital at a meeting of the hospital staff on August 25. Dr. J. E. Ponterio, Shakopee, was named vice-chief-of-staff, and Dr. P. J. Stahler, Jordan, secretary-treasurer.

Windom Hospital, Windom.—Dr. L. L. Sogge, Windom, has been named the first president of the newly formed medical staff organization of Windom

Hospital. Dr. John A. Watkins, Windom, has been made secretary of the group.

Northwestern Hospital, Minneapolis.—Dr. Claude J. Ehrenberg is the new president of the medical staff of Northwestern Hospital. Other officers include Dr. Hewitt B. Hannah, vice president; Dr. Norman E. Rud, secretary-treasurer; Dr. Robert E. Priest, chief of surgery; Dr. Harold E. Miller, chief of medicine; Dr. William P. Sadler, chief of obstetrics and gynecology, and Dr. Donald H. Daniel, chief of general practice. In addition to the officers, members of the executive committee include Dr. Albert T. Hays, with Dr. Cyrus Hansen, radiologist, and Dr. Stanley V. Lofsness, pathologist.

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A new thirty-five bed **convalescent hospital** was opened on September 1 at 2200 Park Avenue, Minneapolis. Manager and director of the hospital is Mr. T. W. Donohue. Dr. Archa E. Wilcox is medical advisor to the hospital.

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An appropriation of $30,000 to maintain a surgical research unit at **Ancker Hospital,** Saint Paul, was approved for 1951 by the Ramsey County Welfare Board and the Saint Paul City Council. The laboratory, which will concentrate primarily on developing heart operations, will be built by private subscription and maintained by the county and city.

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A campaign to raise $168,000 for the construction of a new fifty-bed **St. Francis Hospital** in Shakopee was launched early in September. Getting the campaign off to a good start, a total of $12,000 was pledged by eight physicians in the area.

It is estimated that the completed hospital, fully equipped, will cost about $675,000. The Sisters of St. Francis, who will operate the hospital, will provide $250,000, and a grant from the federal government will provide an additional $303,750.

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Dr. B. W. Mandelstam, administrator of **Mount Sinai Hospital,** being completed in Minneapolis, announced on September 2 that Twin Cities physicians were invited to apply for part-time staff positions at the hospital. He also announced three full-time appointments: Dr. S. Steven Barron, pathologist; Dr. Jack Friedman, radiologist, and Dr. Irving Greenfield, anesthesiologist.

BLUE CROSS-BLUE SHIELD NEWS

More than $5,000 a day including Saturdays, Sundays and holidays is being paid to licensed and registered doctors of medicine by the Minnesota Medical Service, Inc., for services rendered to Blue Shield subscribers. An average of $5,439.65 was paid each of the 243 days between January 1 and August 31, 1950. Each of the 171 work days in the first eight months of 1950 show $7,730.03 going to doctors of medicine for services rendered Blue Shield subscribers. Doctors' services totaling 40,679 were paid during this eight-month period as compared with 21,698, an increase of 19,981 services or 49.1 per cent over the same period of 1949. Excluding Saturdays, Sundays and holidays, the number of checks made ready for mailing each day was 121.

Every effort is being made by the Blue Shield to process doctors' Medical Service Reports promptly so that the physicians will receive their checks in the shortest possible time.

For Your Information

At the end of August, there were 2,691 Blue Shield-participating doctors of medicine in Minnesota. The Blue Shield office is attempting to contact all nonparticipating doctors of medicine, however it would be appreciated if doctors who are at the present time nonparticipating would contact the Blue Shield office relative to enrolling as participating doctors. Also, if you know a colleague who does not participate why not mention it to him for the more participating physicians, the stronger the plan.

To help speed up the processing of Blue Shield cases it would be appreciated if full information could be listed on the reports when first submitted. This information would include the subscriber's group and contract number, the patient's birth date, the diagnosis, type of services rendered the subscriber and other pertinent data. Any information omitted from the report form only tends to delay the processing of that case.

The Minnesota Blue Shield office is receiving an increasing number of requests from licensed and registered doctors of medicine in Minnesota for Blue Shield contracts for themselves and their families. It is regretted that at the present time it is the ruling of the Board of Directors of the Minnesota Blue Shield that physicians cannot apply for and receive Blue Shield contracts. Briefly, the reason is that due to medical ethics physicians tend to render each other medical care on a courtesy basis and to allow licensed and registered doctors of medicine to have a Blue Shield contract would mean that they were using their own organization, Blue Shield, for reimbursement of a service that is ordinarily not reimbursed. Blue Cross, however, is available to doctors in the State of Minnesota and if any physician should desire a Blue Cross contract for himself and his family it is suggested that the request be sent to the Enrollment Department of Minnesota Blue Cross and Blue Shield.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

PRACTICAL GYNECOLOGY. Walter J. Reich, M.D., F.A.C.S., F.I.C.S. Attending Gynecologist, Cook County Hospital; Professor of Gynecology, Cook County Graduate School of Medicine; Attending Gynecologist, Fantus Clinics of the Cook County Hospital; Assistant Professor of Gynecology, Chicago Medical School; Attending Gynecologist and Obstetrician, Grant Hospital; Attending Gynecologist, Fox River Tuberculosis Sanatorium; Consulting Gynecologist, Hazelcrest General Hospital; and Mitchell J. Nechtow, M.D., Associate Attending Gynecologist, Cook County Hospital and the Fantus Gynecologic Clinic; Assistant Clinical Professor of Gynecology, Cook County Graduate School; Associate in Gynecology and Obstetrics, Chicago Medical School; Attending Gynecologist and Obstetrician, Norwegian-American Hospital. 449 pages. Illus. Price $10.00, cloth. Philadelphia: J. B. Lippincott Co., 1950.

RENAL DISEASES. Second Edition. E. T. Bell, M.D., Professor of Pathology in the University of Minnesota, Minneapolis. 448 pages. Illus. Price $8.00, cloth. Philadelphia: Lea & Febiger Co., 1950.

THE PROSTATE GLAND. Herbert R. Kenyon, M.D., Associate Clinical Professor, Department of Urology, New York University, Bellevue Medical Center. 194 pages. Illus. Price $2.95, cloth. New York: Random House, 1950.

CEREBRAL PALSY. By John F. Pohl, M.D., 224 pages. Illus. Price $5.00. Saint Paul: Bruce Publishing Company, 1950.

For quite a number of years a great deal of interest has been focused on cerebral palsy, especially in children. We have witnessed the organization of many groups whose main interest lies in this condition, which claims about half a million victims in the United States. Rehabilitation centers and special schools for handicapped children have sprung up, and many other groups dealing with crippled children have shown increased interest in cerebral palsy, all of which proves the recognition of the importance of this condition and the necessity of using all possible methods for its relief. For these reasons, the appearance of this book is particularly timely and important.

The first chapter states concisely the medical problem. The pathologic anatomy, the different types of cerebral palsy are described, also the diagnostic problems and the evaluation of the mentality. The next chapter goes into the general plan of treatment—muscular relaxation, training of voluntary muscle control and building of developmental patterns. Portions of this chapter deal briefly with drugs, braces, surgical manipulations and outright surgery (brain, cord, nerves, muscles, tendons and skeleton). One chapter is devoted to the important principle of relaxation, while four chapters are needed for neuromuscular training of the various parts of the body. Here we find specific methods to help the patient in gaining voluntary control of muscles by establishing muscle-consciousness, by securing muscle function and co-ordination. The directions are described in detail for the various muscle groups, always complemented by good, instructive photographs. Chapter VIII takes up the training for developmental patterns, taking as an example the progress of such patterns in the normal infant (rolling over, creeping, et cetera) with walking as its ultimate goal (Chapter IX). The last two chapters, which are rather extensive, deal with occupational therapy and speech. The importance of the occupational therapy is very obvious, not only as a means for strengthening and increasing the control of muscles, but as these are "accomplishments for which the child can see a definite purpose, and because they are of immediate usefulness, will attract his co-operation in treatment." The variety of work exercises is remarkable, and these exercises are presented in excellent, clear form. This part includes an important portion on teaching the palsied child to eat by himself and the special eating utensils which are very helpful for the badly handicaped patient. Much space is given to teaching and correcting the speech. This part of the book is most explicit and complete in the discussion of the various defects encountered in these cases and the therapy needed to correct them. As in all the other chapters, many instructive photographs are a helpful feature.

This small volume of 224 pages (which includes an index of 11 pages) is remarkably complete and instructive. The division of the material is good, and each chapter is worked out to such an extent that it gives full information and instruction to anyone interested in this field as a whole or any part of it. The 131 illustrations increase the value of this book considerably. It should be studied most thoroughly by all workers in this field of occupational therapy and speech teachers, but it should also be of great value to physicians and medical students, especially in the branches of pediatrics, orthopedics and neurology. The palsied child owes thanks to the author for the help it will receive through better information and training of his teachers by this work.

R.R.

Classified Advertising

Replies to advertisements with key numbers should be mailed in care of MINNESOTA MEDICINE, *2642 University Avenue, Saint Paul 4, Minn.*

WANTED—Woman Physician to do Obstetrics and Pediatrics, assist older well-established F.A.C.S. Excellent hospital facilities. Salary and percentage from start. Minnesota license or National Boards Parts 1 and 2. Located in suburb of Twin Cities; apartment available. Wonderful opportunity for future. Address E-225, care MINNESOTA MEDICINE.

FOR SALE—Complete modern Westinghouse x-ray equipment, basal metabolism machine, other electrical equipment, instruments, examining table, furniture, et cetera. Will sell at sacrifice for quick disposal. Retiring. Address Charles P. Robbins, M.D., S.W. Corner Third and Center Streets, Winona, Minnesota.

OPPORTUNITY—Newly remodeled physicians' suite suitable for two doctors; fourteen efficient rooms over modern pharmacy on University Avenue, Saint Paul. For information, call Mr. M. J. Collatz, United Properties, Garfield 4303.

WANTED IMMEDIATELY—Young assistant for busy young general practitioner, must have one year internship, rapidly developing industrial community in Northern Minnesota. Salary and commission. House available. Small hospital. Good schools. Address E-229, care MINNESOTA MEDICINE.

WANTED—Young man for permanent position with small clinic group; to do primarily obstetrics. Extra training in this field desirable but not essential. Address E-230, care MINNESOTA MEDICINE.

WANTED—Ophthalmologist, preferably EENT, by well-established South Minneapolis Clinic. Clinic group occupies own new building. For particulars, write E-231, care MINNESOTA MEDICINE.

Mass case finding in hospitals can be effective if applied to two groups—admissions and personnel. It is known that our medical and nursing personnel are only too often exposed to active cases of unknown tuberculosis. This is especially hazardous in the general hospital since the prophylactic nursing techniques usually fall short of those required in a communicable disease institution. The incidence of tuberculosis among doctors and nurses is already several times that of comparable age groups in the general populations, and they should not be needlessly exposed when the method of detection is so readily available.—Hospital Council of Greater New York and Tuberculosis and Health Association, 1950.